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THE PRESIDENT'S ADDRESS*

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For more than three score years my worthy and esteemed predecessors have on similar occasions delivered addresses except when prevented by flood, panic or pestilence. Terminating a year of willing servitude and not unmindful of the honor conferred upon them they have spoken, not to give an accounting of their labor, but on the eve of their departure from office, to give, like the song of the swan, final expression to those things close to their hearts. They have set a noble standard; to emulate it is difficult. The inspiration of their theme was success attained by hard effort; accomplishment had for an honorable price, voicing always hopes for a more glorious future.

I too am proud; I accepted the task determined to follow their precept and example, but unmindful of the danger which surrounds us; unmindful of an awful process of attrition which threatens our high estate, whereby we become no longer masters of ourselves and of our own affairs. The physician, one of the proud handiworks of man, stands insecure in his position, not so proud as his predecessor, possibly to become subservient as the result of his own indifference. Properly to appraise ourselves let's for a moment indulge in retrospect. As we survey the scene, there comes to mind the changing picture of the physician; the transition which has taken place since our forefathers founded this great nation. This period is but a moment in the history of modern medicine, yet history relates that in the colonial and revolutionary days, when men were giants, the physicians were of equal stature; not only in the field of medicine were they supreme, but that in all phases of human endeavor did they take their deserved rank. If that all important human document, the Declaration of Independence were delayed in its borning until now, do you presume that nearly one tenth of its signatures would be those of physicians? We need no answer. For such noble forebears there evolved an American character, a man beloved; closer to the hearthstone than a brother; wise in the ways of the heart, the mind, the soul, and the body; a guardian at birth, a counselor and a confessor in life and frequently a mute witness of the great physician at death. He has disappeared from the scene. We miss him; all mankind misses him. He was the family doctor. Unmindful of facts, we sometimes think of him disparagingly, in that he did not possess the scientific knowledge of today's medicine: the thought contains a modicum of truth, but reveals slight appreciation of this knowledge. It is but natural that each age attribute to itself the acme of all knowledge and achievement, forgetful of the accomplishments of the ages, and that today's truths are born of yesterday's facts and errors. Many of the practices of modern medicine can be traced back through long periods of time, some of them remote. No man can point the finger of derision at a group, among its members an Ephraim McDowell, a J. Marion Sims and a Crawford Long.

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These things are related for a purpose, not to belittle progress in the art and science of medicine, but to point a lesson and adorn a tale. No sensible man would turn back to give up one single triumph of medicine, which has been proved to ease the patient, prevent disease, prolong or save life, but in achieving that progress have we not lost something? In depending upon the biochemist and the gamma ray have we not sacrificed the powers of observation and deduction? Why can we not grasp the one and hold to the other? As we have called art and science to our aid, we have to some extent adopted the artesan too. The art and science of medicine is no longer taught in all cases by one of the art, as manifest in the oath of Hippocrates; we have borrowed from the academic postgraduate school their polished product and welcomed them in our circle. Can we not accept, develop and utilize biochemistry without adopting the biochemist? Human nutrition may have its financial and economic angles but it is essentially a medical field and a medical problem, yet today many of the state and national committees to study and make recommendations on nutrition are dominated by the laity. Medical bacteriology has become the playground of the non-medical graduate and in medical periodicals can be read articles for the physician's consumption, with a hybrid authorship. Today throughout the land the practice of anesthesia has been surrendered almost in toto, the physician no longer collecting the fee, but in most instances it becomes the financial asset of the hospitals. With flashing neon sign and blaring radio we are reminded that the serious function and malfunction of the eye has become the legal domain of one beyond the pale. The hospital boards are predominantly controlled by others and more and more the hospitals are managed by men who have never studied medicine. Not so long ago by legislative act a board was created to direct the public health of this state and the law did not provide that a single physician had to be a member of said board, and but an administration or so ago the state undertook a major hospital set-up throughout the state and not only was organized medicine not consulted but the governor refused to meet its duly elected representative. Since the tragedy of Pearl Harbor we have had the aborted suggestion from the National Government that the medical defense be supervised by social workers, and this same paternal Government in more peaceful times dictates fees for professional work much below, indeed very much below the accepted average over the whole country, with the additional burden of endless paper work on each patient treated. Helplessly we accept from numerous insurance companies a fee schedule much below par, complaining very little and doing nothing about it. Today we face the ridiculous situation of having the hospitals, with which we work hand in hand, entering the practice of medicine under their, as a whole, meritorious group hospitalization schemes, giving medical service to non-hospitalized patients in competition with private physicians and doing the same at a rate below the accepted level. Fearsome is the day when medical care insurance is developed and sold to that point and amount where by force, weight and numbers of policy holders, the insurance companies will dictate to the medical world what it shall receive for its services. The limitation set by law today for the care of the injured employee is frequently an injustice to both the physician and the hospital.

Within our present loose ranks are those who for the sake of temporary honor or financial gain will desert the great majority, selfishness overcoming honor. We witness our own professional brethren using unfair methods of competition and can do nothing about it. Daily we hear in court the testimony under oath with God as the witness of same, giving voice to things which no text ever related or experience taught, the same having weight with the court. This is but a short list of grievances; there is much more evidence that the American physician, once a man standing foursquare, to some extent lord of all he sur-

veyed, is today gradually being subjected to a process of attrition, to a point of helplessness aided and abetted to some extent by quislings within the ranks.

What is the matter? To some extent it can be found in the social tendencies of the times. We cannot expect there will be made a law against it, the usual cure-all; the politicians do not care. Not in an appeal to reason with the public as judge; others have their own problems. Is it to be found in the halls of justice? There, although the generally accepted and acknowledged altruistic purposes of medicine are recognized as are the doctors' enormous contributions to charity and freely given aid to our fellowmen, we are condemned and found guilty. In that pronouncement is pointed out the way to fulfill the biblical admonition: "physician cure thyself." The way is clear. Only by a strong organization swinging to the left, bringing force to bear when needed, ever maintaining our high ethics and ideals to the point of suffering for our rights and if necessary temporarily losing our liberty, can we again be self respecting men, dictating medical affairs which is our privilege and our earned right. It is my earnest conviction that what is good for organized medicine is good for the nation, the state and the race. We can only rule in the present state of society by an organization that is strong in nature and powerful to enforce its mandates, call that organization by any name you please. The democracies felt secure; libery is in danger. If we passively wait we will lose even that which we still retain.

PRACTICAL ASPECTS OF WATER BALANCE

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Water comprises 70 per cent of the body weight. All of the water in the body is free, none in chemical combination with other substances. About 75 per cent of the body water is in the cells, about 20 per cent is in the tissue fluids, and about 5 per cent in the blood plasma. The body tends to keep its water content constant, so that variations in intake of water produce corresponding variations of output. The body has an even greater tendency to keep its intracellular water constant, so that variations in total body water take place largely at the expense of the tissue fluids and the blood plasma.

The sources of body water are fluids ingested as such, free water contained in solid and semisolid foods, and water produced in the body by the combustion of foodstuffs. Under ordinary circumstances the largest single source of body water is the free water in solid and semisolid foods.

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Water is lost from the body in the urine, the expired air, from the skin, and in the feces. Under ordinary circumstances the amount lost in the urine is greater than that lost by any other route.

Under normal circumstances there is a perfect balance between water intake and production on the one hand, and water output on the other. Under average conditions the balance among various sources and losses of water per day is about as follows:

Intake and Production		Loss	
Liquids as such	1000 c. c.	Urine	1500 c. c.
Free H_2O in foods	1200 c. c.	Sweat	500 c. c.
Water of metabolism	300 c. c.	Expired air	350 c. c.
		Feces	150 c. c.
Total	2500 c. c.	Total	2500 c. c.

If environmental conditions remain constant, particularly environmental temperature, variations in the total intake of fluid are manifested principally by corresponding variations in the output of urine. Healthy persons will remain in water balance under ordinary environmental temperature if the total available water is reduced to as low as 1500 c. c. daily, so that a healthy person will get along for a time at least if he takes no fluid at all as such, but

consumes a normal amount of solid and semisolid food. Under such a circumstance his urinary output would be only about 500 c. c. a day; if his kidney function is normal, adequate excretion of all urinary constituents would be attained in this small volume, but kidney damage would eventually supervene.

If the intake of available water is further reduced, water balance is maintained for a time by a further reduction in the output of urine and by a diminution in sweating and the fluid secreted by the glands of the gastrointestinal tract. An individual under such a circumstance would have a dry skin and dry tongue without actually being dehydrated. However, because of the greatly reduced output of urine, urinary constituents would accumulate in the blood, and the patient might die of uremia without extreme dehydration of his body tissues. If any other factor supervened which would prevent conservation of fluid by the kidneys and gastrointestinal tract, such as polyuria due to diabetes, or vomiting, or diarrhea, marked dehydration would occur, which would be manifested by softness of the eyeballs and looseness of the skin. Death under these circumstances might be due to a combination of dehydration and uremia, or even to dehydration alone.

A healthy person, conversely, will remain in water balance if his intake of fluid is tremendously increased, provided other factors, especially his intake of sodium, remain unchanged. Thus he can take as much as 3,000 to 4,000 c. c. of water by mouth in a single day and compensate readily by an increased output in urine. If a large amount of water, as for example a quart, is taken at one time, there is a delay of about an hour during which no diuresis occurs, and then suddenly over a period of an hour or less, almost the exact amount of fluid ingested is excreted by the kidneys. The mechanism of this water diuresis is not understood. Immediately after the water is taken, an increase in blood volume occurs, but the blood volume returns to normal before diuresis begins. However, under certain circumstances which will be discussed later, the fluid output of the body is less than the intake, and water accumulates in excessive amounts in the tissue spaces. If the accumulation is gradual, it is manifested at first by a gain in weight; when the amount retained exceeds eight or ten pounds, discernible edema is present. In extreme retention fluid accumulates in the body cavities, edema of the lungs sets in and death by "drowning" occurs.

Unfortunately water balance is not a simple balance between intake and output. Water balance is intimately linked with electrolyte balance and thus with acid-base equilibrium, and also with the blood pressure in the capillaries, with the permeability of the capillary walls to colloid particles, with the concentration of the proteins of the blood plasma, and with the secretion of certain hormones.

The most abundant cation of the tissue fluids and blood plasma is sodium, and the most abundant anions are chloride and bicarbonate. There is very little sodium in the cells; in these potassium is the principal cation. Cell membranes are not freely permeable to cations; hence the intracellular fluid is always low in sodium and the tissue fluid and blood plasma are always low in potassium.

The principal source of body potassium, sodium, and chloride is from ingested food, while bicarbonate is a product of body metabolism. Under normal circumstances the concentration of electrolytes in both the cells and body fluids is kept constant through the elimination or retention of water; also the relationship of cations to anions is kept constant by the retention or elimination of these substances in the urine, a process which affects the water balance. Thus the ingestion of large quantities of sodium chloride tends to cause retention of water in the body, so that the electrolyte concentration of the tissue fluids may remain constant. The increase in body water occurs solely in the tissue fluids; the plasma volume would tend to increase, but this tendency is counteracted by other factors which maintain blood volume. Similarly,

complete deprivation of salt results in a loss of body water at the expense of the tissue fluids. Loss of chloride without sodium, as in the vomiting of gastric juice, results in increased execretion of sodium in the urine so that a constant anion-cation ratio is preserved; water is required for this excretion, so that a net loss of body fluid results. The occurrence of acidosis due to any cause results in the urinary excretion of acid sodium salts, and thus leads to loss of body water.

Of great importance in water balance is the exchange of water between blood plasma in the capillaries and the fluid in the tissue spaces which surround them. This exchange is normally accomplished by the hydrostatic pressure of the blood within the capillaries, which tends to force fluid out through the capillary walls, and the osmotic pressure exerted by the plasma proteins, which tends to "suck" water into the capillaries. Under normal circumstances these counteracting forces are delicately balanced, so that near the arterial end of the capillary the fluid passes out, while near the venous end it is drawn in. This balance may be disturbed by three factors, each of which would tend to cause loss of fluid from the capillaries into the tissues, and thereby cause retention of water in the body. The first of these is increase of the blood pressure within the capillaries; this is usually due to heart failure or venous obstruction. The second is depletion of the plasma proteins, which may be due to starvation, liver disease, or loss of protein in the urine. The third is damage to the capillary walls, so that they become permeable to protein which escapes into the tissues and thereby lowers the effective osmotic pressure of the blood; this occurs in an outstanding form in surgical shock, in which a large portion of the blood plasma escapes suddenly into the tissue spaces. This state of affairs also probably obtains in acute nephritis.

Water balance is also affected by the antidiuretic hormone of the posterior pituitary and the steroid hormones of the adrenal cortex and the gonads. A posterior lobe hormone, probably elaborated under the influence of the hypothalamus, inhibits the excretion of water by the kidney under normal circumstances; when this hormone is deficient, tremendous diuresis occurs, much like the diuresis due to ingestion of large quantities of water. In such an event water balance is maintained by a greatly increased fluid intake, and electrolyte balance is not disturbed. The antidiuretic hormone of the pituitary is probably concerned in the diminution of the urinary output which occurs when the fluid intake is reduced in normal persons, or when fluid is lost from the body due to vomiting or diarrhea or marked sweating. There is some evidence that secretion of excessive amounts of this hormone may play a role in the production of edema in the nephrotic syndrome.

The steroid hormones of the adrenal cortex and gonads cause retention of sodium in the body and thus favor the retention of water. Synthetic steroid hormones, such as stilbestrol and desoxycorticosterone, have the same effect. Physiologic and pathologic variations in the quantities of these hormones secreted affect water and electrolyte balance considerably. Premenstrual edema, for example, is probably due to sodium retention resulting from the effect of progesterone. In Addison's disease, deficiency of adrenal cortex hormones leads to marked loss of sodium and thus to dehydration.

Vitamins also affect water balance indirectly. The most important of the vitamins in this regard is thiamin, the lack of which leads to cardiac insufficiency with resultant increase in the hydrostatic pressure within the veins and capillaries.

RULES AND PRINCIPLES GOVERNING WATER BALANCE

It is thus clear that water balance depends not only on the intake and loss of water *per se*, but also upon intake of food, the intake and loss of electrolytes, the acid-base balance, the integrity of cardiac and kidney function, and upon other factors as well. It is also clear that the maintenance of fluid balance in the treatment of disease is not a simple matter of giving enough but

not too much water. In most cases, however, it is largely a question of how much water and salt to give, as the various factors concerned in retention and loss of water must be considered, and routes of administration of fluid are also important. The following are general rules and principles which take into consideration most of the factors which have been discussed, and which may be applied to most of the problems of water balance.

1. In the absence of factors which cause excessive fluid or electrolyte loss, an average adequate intake of fluid for a patient who can take an average diet is about 1000 c. c.

2. The less solid food taken, the more fluid as such is required. On a "full liquid" diet, the average requirement is about 2000 c. c., and if nothing is given by mouth the requirement is about 2500 c. c. Electrolyte balance need give no concern if a reasonable quantity of food and fluid can be given by mouth, but if all liquids are given parenterally salt and glucose must be supplied, and vitamins and protein must be given (thiamin, nicotinic acid, ascorbic acid, blood plasma or whole blood) if it is necessary to continue parenteral feeding for more than a few days. A patient can ordinarily be kept in fluid and electrolyte balance and in a fair state of nutrition for several days if 1000 c. c. of 5 per cent glucose in normal saline and 1500 c. c. of 5 per cent glucose in distilled water are given daily. This supplies 2500 c. c. of water, 8.5 grams of salt, and 600 calories.

3. If excessive fluid and electrolyte loss has occurred or is occurring, extra fluid and salt must be supplied, the amounts being gauged largely by careful observation of the patient and by measurement of the urinary output. Estimation of the approximate amounts of fluid and electrolyte lost by other routes is of some value, but it should be remembered that such estimates are rarely accurate. The value of determination of the CO_2 combining power of the blood plasma and the plasma chlorides is limited because of the marked tendency of the body to maintain a normal concentration of electrolytes in the blood plasma in spite of great reduction or increase in the total amounts of these substances in the body.

4. The common causes of excessive fluid and electrolyte loss, and thus the common causes of dehydration, are vomiting, diarrhea, atony of the gastrointestinal tract, excessive sweating, and diuresis.

5. The common causes of excessive fluid and electrolyte retention, and thus of overhydration are the excessive administration of water and salt or salt alone, heart failure, nephritis, nutritional deficiency, administration of steroid hormones, marked albuminuria due to any cause, and toxic and allergic states which may affect capillary permeability. Next to heart disease and nephritis, perhaps the commonest cause of overhydration is overtreatment of dehydrated patients.

6. The signs of dehydration are oliguria, looseness of the subcutaneous tissues, dryness of the tongue, and in extreme cases softness of the eyeballs and stupor or coma. In certain cases the CO_2 combining power of the blood plasma and the plasma chlorides are altered, and the relation of these determinations one to the other may give important clues as to the causes of the dehydration. These determinations, if made upon the same specimen of blood, constitute an accurate index of the *concentrations* of sodium and chloride in the blood plasma and tissue fluids, but give no inkling at all as to the total *amount* of these substances in the body. Nearly always when dehydration is present both sodium and chloride as well as water are needed, irrespective of the cause of the dehydration. The most reliable sign of incipient dehydration is diminution of the output of urine.

7. The signs of overhydration are increased tension of the subcutaneous tissues, puffiness of the face, and in extreme cases generalized edema, with dyspnea due to pulmonary edema and hydrothorax. The output of urine is usually reduced, but it may be normal, rarely high. The most reliable sign of early overhydration is increasing weight; when this factor cannot be deter-

mined puffiness of the face is the earliest definite sign. Overhydration always means an increase of total body sodium, and therefore of chloride as well; it thus calls for temporary complete interdiction of salt, as well as for restriction of water.

8. Normal water balance is manifested by a normal output of urine (1200-2000 c. c.) daily, by normal tension of the subcutaneous tissues, and by normal moisture of the tongue. A daily urinary output in excess of 2000 c. c. may mean that too much fluid or too little salt is being given. Generally such an occurrence, if the patient looks well, calls for slight decrease in fluid and increase in salt intake. If signs of dehydration coexist with a large urinary output the intake of salt should be increased. If signs of overhydration coexist with a large urinary output the intake of salt should be decreased. Normal water balance is easily maintained in the presence of normal cardiac and renal function if one measures the urinary output and watches for early signs of overhydration and dehydration.

9. Impairment of kidney function increases the difficulties in maintaining water balance, and renders the individual more susceptible to dehydration and overhydration. Generally speaking, moderate or marked impairment of kidney function is accompanied by compensatory polyuria which may predispose to electrolyte and water depletion; hence in such cases more salt and water are needed. However, the ability of the damaged kidneys to excrete sodium and chloride is impaired, so that salt retention and consequent overhydration occur if the optimal salt intake is exceeded. Even in the presence of complete anuria overhydration will not be produced, nor dehydration corrected, if salt is withheld completely. The usual principles of maintaining normal hydration apply in cases of impaired kidney function, but more careful observation, which should include NPN determinations, is indicated. Generally speaking, considerably more water and a little more salt are needed in order to maintain an adequate urinary output and normal water content of the tissues.

10. Particularly difficult to treat are patients in whom both the cardiac and kidney functions are impaired. The tendency in these patients is toward overhydration, with a diminished output of urine and nitrogen retention. Extremely careful observation is necessary, which should again include daily determination of the blood non-protein nitrogen. Generally speaking, fluid should be forced in order to promote a large urinary output, and salt restricted so as to prevent the formation of edema. Too drastic salt restriction may result in dehydration even though large amounts of fluid are given. The aim is to restore normal hydration of the tissues and a normal level of the non-protein nitrogen of the blood.

11. If the gastrointestinal tract is functioning normally, fluids and food should be given only by mouth. Tube feeding is preferable to parenteral feeding in unconscious or delirious patients. When fluid and electrolytes and food must be given parenterally, the intravenous route is usually preferable to the subcutaneous. In extreme dehydration, which has led to diminution of the blood pressure and of the pulse pressure, fluids given by any other route will not enter into the circulation.

PRACTICAL APPLICATION OF RULES AND PRINCIPLES

Finally, the application of these rules and principles in a few specific types of cases in which water balance is a problem, may be considered.

I. *Acute febrile illnesses*, such as pneumonia, influenza, and typhoid fever. In these conditions there is a variable increase in the loss of fluid by other routes than the kidneys—by the lungs or skin or the gastrointestinal tract, and if the caloric intake and intake of solid food must be reduced there is a decrease in available water not taken as liquid *per se*. The indication, therefore, is for a sufficient increase in the intake of liquids as such to maintain a normal urinary output. Ordinarily in cases of pneumonia the daily intake of fluid need not

exceed 2000 or 2500 c. c. In typhoid fever the daily requirement may be slightly less if a high caloric soft diet can be given. In pneumonia electrolyte balance will be easily maintained by the salt in soups and milk, and it is certain that for the typhoid patient adequate salt will be available in the diet. Overhydration may occur during the latter weeks of typhoid fever if inadequate amounts of protein and vitamins are supplied.

II. *Diabetic acidosis:* In this condition dehydration is produced by several factors; the diuresis of uncomplicated diabetes, the loss of sodium in the urine as acid sodium salts, vomiting, sweating, and the inability to take fluids by mouth. The electrolyte stores of the body are tremendously depleted. The concentration of chloride in the blood plasma and tissue fluid is usually normal, but total body chloride is markedly reduced. Even the concentration of sodium in these fluids is markedly reduced, as is indicated by the low CO_2 combining power of the blood plasma. The special need of the body in order for water balance to be restored is for water, sodium, and chloride. Acid-base balance will also be restored by supplying water and salt if kidney function is good, because the first tendency of the body will be to restore normal concentrations of electrolytes and the early excess of chloride ions will be promptly excreted in the urine. If the acidosis is severe or of long duration, and especially if the administration of saline does not produce prompt diuresis, sodium bicarbonate or lactate rather than the chloride should be given, lest in such a case an increase in total body base may not correct the disproportion between the concentrations of acidic and basic radicles. The indication, therefore, is to give, intravenously, a large volume of saline and perhaps a smaller amount of sodium bicarbonate. I would recommend giving 1000 c. c. of normal saline over a 30 minute period, and then continuing its administration more slowly, 40 to 60 drops a minute, until the patient can take and retain fluid by mouth. There is no harm in giving glucose with the saline, and its employment obviates the danger of hypoglycemia occurring before acidosis has been corrected. If the output of urine remains small after the first 1000 c. c. of saline has been given, it is advisable to give 500 c. c. of 5 per cent sodium bicarbonate solution. Insulin, of course, should be given, and if there are signs of marked circulatory depression (thready pulse) it should be given intravenously rather than subcutaneously. It should be emphasized, however, that water, salt and sodium bicarbonate directly combat the altered physiology of diabetic acidosis, while insulin acts indirectly and more slowly, by diminishing the necessity for the oxidation of fat. It is also important to remember that in advanced diabetic acidosis true shock may exist, and that saline alone will not restore blood volume. If the pulse volume does not improve promptly after the administration of saline has been started, blood plasma or whole blood should be given.

III. *Severe mercury poisoning with anuria:* In such a case there is apt to be dehydration in spite of anuria, due to the loss of water and electrolytes in vomiting and diarrhea. Both CO_2 and chlorides will be low. The non-protein nitrogen of the blood rises steadily from day to day. The indication is to maintain water and electrolyte balance as near to normal as possible, for it has been established that persons survive anuria for much longer periods if electrolyte depletion can be prevented. Overhydration must also be avoided, for it is easy to "drown" anuric patients. During the first few days it is advisable to give glucose and saline continuously by the intravenous drip, in the amount of about 5000 c. c. daily; this will certainly overcome fluid and electrolyte depletion, but also will certainly lead to overhydration within a few days. As a matter of fact, edema may occur when the concentration of sodium in the blood plasma is still below normal. The proper procedure, therefore, is to watch carefully for the earliest signs of overhydration, and when these appear to omit saline in the infusions and to reduce their total daily amount to about 1500 c. c. When

the signs of overhydration have disappeared, saline should again be given, the amount from day to day varying with the signs of dehydration or overhydration. Careful regulation of the amount of salt and water given may enable one to tide the patient over until the secretion of urine is reestablished.

IV. *Protracted vomiting in heart failure with edema:* If it can be established that the vomiting is not due to digitalis intoxication or to organic disease of the gastrointestinal tract, one may assume that the vomiting is due to edema of the abdominal viscera. The patient, therefore, is overhydrated, and from the standpoint of water balance alone the vomiting is probably not harmful. However, the vomiting is harmful because it prevents the absorption of food and vitamins. The treatment should be directed toward improving cardiac function and toward mobilizing and removing the edema fluid. Ascitic fluid should be removed by tapping. Diuretics should be employed. One should be sure that digitalization is adequate. Such food and fluid as can be retained should be free of salt. Vitamins should be given parenterally. Calories may be supplied by giving as much as 2000 c. c. of 10 per cent glucose solution daily by very slow intravenous infusion, or a smaller volume of a more concentrated solution may be given. The administration of fluid without salt will not increase the edema.

SUMMARY

Although many factors are concerned in the balance between fluid intake and output, water balance can usually be maintained or restored by careful regulation of the amounts of water and salt which are given to sick persons. Measurement of the output of urine and the physical examination of the patient yield the most reliable signs of dehydration and overhydration, and usually indicate the need for increase or decrease in the amounts of water and salt which should be given.

PEPTIC ULCER

REPORT OF AN UNUSUAL CASE

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The average patient with an uncomplicated peptic ulcer presents the ordinary therapeutic problems; complications bring the internist and surgeon into debate and mortality into consideration. It is not unusual to have a refractory instance, nor is it rare to find more than one complication in a single individual. It is commonly assumed that the patient who experiences misfortunes, despite accurate therapy, is most likely one of the constitutionally predisposed persons.

When one encounters a patient so inherently susceptible to ulceration and its complications, it is to be wondered then why the average patient will respond somewhere along the therapeutic gauntlet. The case we herewith present defied all percentage for complications, survived more than statistics permit and on the whole proved the "so-called" ulcer diathesis is extremely difficult to cope with medically and surgically.

CASE REPORT

A. W. was an introspective 36 year old Jewish male who, at the age of 18 manifested clinically and roentgenologically a duodenal ulcer. He obtained symptomatic relief from temporary adherence to a modified Sippy regimen. On May 20, 1925, when 20 years old, he developed an acute (afebrile) abdomen for which an emergency cholecystectomy and appendectomy were done. The surgeon's notes included: "the gallbladder was adhered to the duodenum, pulled forward and to the right." The pathologic report revealed a normal gallbladder and appendix. Symptoms persisted postoperatively, but were relieved by a medical ulcer regimen.

On April 8, 1929, he was rehospitalized with severe ulcer pain complicated by obstructive manifestations; he was relieved by an acceptable bed rest ulcer routine. Eighteen months later (October 11, 1930) recurrent pain and obstructive symptoms brought him back to the hospital and an anterior gastroenterostomy and enteroenterostomy were

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done. (An anterior gastroenterostomy was elected because of technical difficulties.) After a two month interval (December 2, 1930) he was readmitted with an ulcer roentgenologically demonstrable on the gastric side of the stoma. Medical therapy alleviated the symptoms. Another admission, March 5, 1931, recorded the successful medical management of melena.

Acute abdominal pain indicated hospitalization on April 29, 1931, and an exploratory laparotomy revealed a perforated gastric (marginal) ulcer which was excised and the area plicated. Recurrent ulcer pain in 1933 and in 1934 were controlled by a well-conducted ulcer regimen; duodenal feedings (by tube) were used for two weeks on one occasion.

The patient was not hospitalized again until September 8, 1935, when he returned with an acute gastric perforation developing subsequent to severe obstructive manifestations. A subtotal gastrectomy was successfully performed.

On December 5, 1935, he was readmitted after a severe hemorrhage which was controlled medically.

Two months later (February 18, 1936), a so-called "subacute perforation" was managed medically, but three days later he had an acute perforation of a marginal ulceration. An extensive gastritis and jejunitis were apparent at exploratory with an inflammatory mass on the gastric side of the anastomosis palpably and macroscopically suggestive of malignancy to the surgeon and surgical pathologist. A subtotal gastrectomy and resection of the involved area of the jejunum was successful. (Microscopic pathology proved the lesion benign.)

On June 8, 1936, after an asymptomatic period, the patient had a severe acute hematemesis and was admitted to the hospital in shock with a red blood cell count of 2,250,000. He responded to a conservative regimen.

One month later, having had persistent severe pain, an alcohol injection of the twelfth thoracic and first lumbar sympathetic ganglion, including the sphlanchnics, was resorted to with relief.

He remained under close observation for the next twelve months with postprandial discomfort persistent and gastric analysis repeatedly approximating a free hydrochloric acid of 70 units. In 1939 and 1940 he was entirely asymptomatic and when called in for a follow-up examination in October 1941, he was and had been in excellent health, was not adhering to any regimen, smoked to excess and was drinking freely. He had gained 45 pounds during the four year period. Physical examination revealed an apparently healthy 36 year old male. Gastric analysis showed an achlorhydria with total acids of 9 units. Roentgen study revealed a coarse mucosal pattern, a well functioning anastomosis with some sphincter action; there was no demonstrable lesion.

SUMMARY

There are numerous unusual factors in this case report.

1. The patient may be classified as a "juvenile" at the onset of his illness, being only 18 years old when a duodenal ulcer was proved roentgenologically.

2. All of the complications other than malignant change occurred in his instance: (a) perforations: gastric, jejunal and anastomotic; (b) hemorrhage: on four occasions; (c) multiple ulceration: duodenal, gastric, anastomotic marginal (gastric) anastomotic or marginal (jejunal); (d) obstruction: duodenal, anastomotic; (e) intractable pain.

3. Extensive gastric surgery was survived: (a) anterior gastroenterostomy; (b) partial gastrectomy; (c) excision of ulceration and plication; (d) subtotal gastrectomy.

4. Symptoms persisted until an achlorhydria occurred.

ACUTE PERFORATION OF PEPTIC ULCER

REPORT OF 29 CASES

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The present communication is an analytic report of the experience with the various factors dealing with the subject of acute perforated peptic ulcers as it obtains at the Lafayette Charity Hospital. It is based upon a study of 29 cases collected from June, 1938, through June, 1941. Every case but one was submitted to a surgical procedure and the diagnosis confirmed. Operations were performed by six different members of the staff.

The general management of the patient was the same in all instances with but slight modification in the more seriously sick. As soon as admitted, the patient was seen by the admitting officer who determined the advisability of having x-rays taken before being moved to the ward. Transfusion or

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intravenous infusion was given while preparations for operation were made. Neoprontosil was administered parenterally in most patients. In those individuals who were exceedingly ill or the danger great, x-rays were not taken, but they were immediately transported to the operating room where all medication and preparation was carried out. None of the patients seen, in whom perforation had occurred within 12 hours, appeared in shock as determined from the pulse, temperature, respirations or blood pressure readings. The abdomen, however, still retained its almost board-like rigidity.

Of the total number of cases, 29, only one occurred in a white female. This patient was admitted in coma. A period of 67 hours had elapsed from the onset of her illness until her admission. In this case the diagnosis was made by the x-ray findings of air under the diaphragm. She was not operated upon. This was the only female in the series. Of the males, there were 18 white and 10 colored. This is in approximate proportion to the number of white and colored admissions, that is 60 per cent white and 40 per cent colored; 62.0 per cent white perforated and 38 per cent colored perforated. Of the patients who died following operation, eight were white and four colored, which is in proportion to the admissions and incidence of disease.

The gross mortality of the entire group was 37.9 per cent; 18 patients recovered and 11 died. This includes a white woman who was admitted 67 hours after the onset and when admitted was in coma, showing a 4 plus glycosuria and who was not operated upon. Of the others, two were in extremis when admitted but were nevertheless submitted to operation. The exclusion of these three cases would have reduced the mortality rate of 27.5 per cent, demonstrating again how fickle statistics may be.

The interval of time that elapses from the moment of perforation until the patient reaches the operating table has long been known to be the most important factor affecting survival. An average of this time interval in the patients who survived in this series was 10 hours. In the cases that ended fatally, the average time interval was 28½ hours.

Drainage has always been a problem. In this series, no drains were placed in seven patients, or 28 per cent. Of this group, five lived and two died. The time interval in the group that survived and not drained varied from one to 13 hours after the onset of symptoms. Of the two who died, the time factor was 10 and 48 hours respectively. The question of drainage was not a preference by any one surgeon but was evenly distributed and therefore represented the impression of the gravity of the individual case by each surgeon. In a review of the literature it would seem as if the more serious the case the more drains were placed. However, it does seem that better results are obtained in not draining when the general condition of the patient is good and the risk slight. This rule was not applied in this series. Drainage was influenced chiefly by the amount of fluid present in the peritoneal cavity. Of those who survived, drainage was performed in 61.5 per cent and in those who died, in 80 per cent.

The age incidence of perforated peptic ulcers is similar to that of the uncomplicated ulcers. The greatest decade incidence in this series for both white and negro was from 21 to 30.

AGE AND COLOR INCIDENCE		PER CENT	WHITE	COLORED
0-10	0	....	0	0
11-20	1	4	0	1
21-30	10	34	7	3
31-40	7	25	3	4
41-50	8	28	6	2
51-60	1	4	1	1
61-	1	4	1	0

The incidence as to seasons is very interesting. Only one case occurred in the summer quarter. The spring quarter contained the highest number of cases with May as the greatest month. This observation is not new and has provoked many unusual explanations. The most persistent theory is the one stating that the occurrence of more vitamin containing foods at this season served as a protection. Inasmuch as 50 per cent of these patients were farmers to

whom the same vitamin foods should have been available for a longer period than the summer quarter, it would appear as if some other factor was present in this series.

It has occurred to me that at this time of the year, one is apt to eat less food and that food is more likely to be concentrated and more easily digestible. Furthermore, the residents in this area are accustomed to very rich and highly seasoned foods and these seem to be avoided in the summer months.

SEASONAL INCIDENCE

January	4	July	0
February	1	August	1
March	2	September	0
	7		1
April	4	October	3
May	8	November	2
June	2	December	2
	14		7

In the majority of these cases, in spite of careful questioning, no history of previous gastric complaint could be elicited. Nevertheless when the abdomen was opened, the pathologic condition about the ulcer suggested the presence of an ulcer for some time. It is possible that the pain sensitivity level of this group of patients was such that the usual symptoms of peptic ulcer were either ignored or tolerated without much concern. One point is definite, the sudden pain of rupture is severe enough to all to demand immediate attention. The site of perforation in these individuals was either on the anterior surface of the stomach or of the duodenum.

SUMMARY

While the series of cases reported is not great, it does bring out certain pertinent factors which operate in this section of the state:

1. The presence of 29 cases of perforated peptic ulcers within a period of three years in a general charity hospital of 220 beds is a large number.

2. The ratio between the races indicates that there is no species susceptibility.

3. The gross mortality of 37.9 per cent is very high and in this series points to a delay on the part of the family or the physician in presenting the patient for operation, or diagnosis.

4. As in other reports, the more seriously ill were more apt to be drained than those in better condition. In this report, it mattered not as the results were not affected by anything but the time it took to get the patient to the operating table.

5. The greatest age incidence was the decade between 21-30.

6. The highest number of perforations were admitted in May and the lowest seasonal period was the summer months. As suggested, this is the period when the least amount of food is consumed and condiments added are at the lowest point.

7. Ulcers on the anterior wall of the stomach or duodenum perforate suddenly and the only treatment is surgical.

LEFT-SIDED APPENDICITIS

A REPORT OF THREE CASES

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AND
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The three cases of left-sided appendicitis reported in this paper were found in a series of 4215 appendectomies performed in a twenty year period, 1921-1940, by James Thomas Nix, M. D. During this period, four patients with situs inversus and complete rotation of the colon were seen in the course of treatment for conditions unrelated to the appendix. One of these patients had previously been operated on for left-sided appendicitis.

The study of cases and review of the literature is made by James Thomas Nix, Jr., M. D.

Embryology of the Appendix: In the third month of intra-uterine life, the cecum and appendix rotate from the left to the right iliac fossa.

Classification of Left-sided Appendices: According to Hembrow,[3] left-sided appendices fall into four categories: (1) complete or incomplete visceral transposition; (2) non-rotation of colon; (3) imperfect fixation of the cecum and ascending colon;

(4) excessively long appendix with the tip to the left of the mid-sagittal line.

Classification of Left-sided Appendictis: A left-sided cecum and appendix may give clinical signs in the right or left lower quadrant. Hembrow believes that appendicitis in cases of: (a) non-rotation of the colon; (b) imperfect fixation of the cecum, and (c) excessively long appendix with the tip to the left of the mid-sagittal line should show clinical localization in the right lower quadrant of the abdomen. In complete visceral transposition, the clinical signs should be on the left according to Hembrow,[3] on the right according to Block,[1] on either side according to Pol.[5] If repeated attacks of appendicitis result in adherence of the appendix to the left side of the abdominal wall, the clinical signs should be on the left.

Incidence of Left-sided Appendices: Le Wald[4] found one case of complete transposition of the viscera in 5000 necropsies; 29 cases of complete transposition of the viscera in 40,000 roentgen ray examinations (1:1400); and nine cases of non-rotation of the colon in 40,000 roentgen ray examinations or one case in 4400.

Incidence of Left-sided Appendicitis: Left-sided appendicitis, according to Walther, occurs once in every one thousand cases. Block[1] states: "Pol in 1935 found 46 cases reported of left-sided appendicitis when a situs inversus viscerum was present; totally in 34, partially in 12."

Diagnosis: In the 45 cases reviewed by Pol,[5] correct preoperative diagnosis was made only in seven. Left-sided appendicitis must be differentially diagnosed from: (1) right-sided appendicitis; (2) diverticulitis, (3) salpingitis, and (4) ectopic gestation.

CASE NO. 1

INCOMPLETE VISCERAL TRANSPOSITION

Miss J. K., a white female, 25 years of age, was admitted to Hotel Dieu, May 3, 1921. Patient complained of diffuse abdominal pain, nausea, and vomiting. Physical examination on admission showed the abdomen to be diffusely rigid with tenderness on light palpation; examination of the chest showed PMI in left fifth interspace; no dextrocardia; no abnormalities.

The diagnosis of ruptured appendix with diffuse peritonitis was made and an emergency appendectomy was performed under ether anesthesia. A low gridiron incision was made in the right lower quadrant of the abdomen. Free purulent fluid was found in the peritoneal cavity. No large bowel was found in the right hemiabdomen. A corresponding gridiron incision was made in the left lower quadrant of the abdomen. The cecum appeared in the incision. The appendix, gangrenous and perforated at the tip, was removed.

After an uneventful postoperative course, the patient was discharged from the hospital cured, May 26, 1921.

(Records on this patient are meagre, hospital charts for this period having been destroyed by flood.)

CASE NO. 2

IMPERFECT FIXATION OF THE CECUM AND ASCENDING COLON

Mr. P. B., a white male, 23 years of age, was admitted into Hotel Dieu, September 22, 1929, complaining of diffuse peri-umbilical pain with nausea and vomiting. The patient gave a history of intermittent aching pains in the epigastrium, unrelated to the consumption of food, for about five months' duration. On the night before admission, the patient had eaten a plate of raw oysters. Physical examination showed PMI in left fifth interspace, no dextrocardia, no abnormalities of the chest; the abdomen presented no rigidity or distention, but a slight tenderness in left lower quadrant of abdomen. A tentative diagnosis of acute gastroenteritis was made.

The three blood counts made on the day of admission showed (1) total white cell count of 12,250, small lymphocytes 12, large lymphocytes 0, neutrophils 88; (2) total white cell count 14,500, small lymphocytes 8, large lymphocytes 3, neutrophils 89; (3) total white cell count 19,750, small lymphocytes 7, large lymphocytes 3, neutrophils 90. The blood count on the second day of hospital stay was: total white cells 8,000, small lymphocytes 21, neutrophils 79. Repeated urinalyses were negative. Patient was discharged on the second day of his hospital stay.

On October 7, 1929, patient was readmitted for an interval appendectomy. The physical examination on this admission showed no distention, no rigidity and no tenderness of the abdomen. Repeated urinalyses were negative. An operation lasting one and a half hours was performed under ethylene anesthesia. A low gridiron incision was made in the right side. Upon opening the peritoneum, no evidence of the large bowel could be seen. A gridiron incision in the left lower quadrant revealed the sigmoid to be normally located. The cecum was mobile, situated to the left of the midline. The appendix was removed. The patient recovered with a smooth postoperative course and was discharged from the hospital October 21, 1929.

The pathologic report was as follows: The appendix, 5½ cm. in length, was of normal diameter. The outer surface was injected and presented numerous fibrous tags. Diagnosis: subacute appendicitis.

CASE NO. 3

INCOMPLETE VISCERAL TRANSPOSITION

Miss B. B., a white female, 12 years of age, was admitted into Hotel Dieu, June 20, 1939; patient complained of diffuse aching pain in the epigastric region, nausea, and vomiting. Physical examination showed PMI in left fifth interspace, no dextrocardia, no abnormalities of chest; no distention or rigidity of the abdomen; tenderness to light palpation in the epigastrium.

The blood morphology made on the day of admission showed: total white cell count 9,500, neutrophils 70, lymphocytes 30. The blood count made later on same day showed: total white cell count 11,000; lymphocytes 25; neutrophils 73; metamyelocytes 1; staff 1; segmented 71; eosinophils 1; basophils 1; red cell count 3,600,000; hemoglobin 55 per cent; color index 0.8. The blood count made on the second day of the hospital stay showed: total white cell count 7,500; lymphocytes 21; neutrophils 79; staff 2; segmented 77; eosinophils 1; red blood count 4,450,000; hemoglobin 70 per cent, color index 0.8.

Urinalysis made on the day of admission showed acetone 2 plus and occasional pus cells.

The patient was operated on 48 hours after admission under cyclopropane anesthesia, the operation lasting one hour. Through a low right gridiron incision the abdomen was opened. The sigmoid was present on the right side. A counter incision was made on the left. The cecum was located on the left side with the appendix pointing toward the midline. The appendix was removed. The patient recovered with a smooth postoperative course and was discharged from the hospital on July 3, 1939.

The pathologic report was: The appendix, 10 cm. in length and ½ cm. in diameter, presented thickened walls, covered with fibrous tags. The diagnosis was chronic appendicitis.

INTERPRETATION

The incidence of left-sided appendicitis in this series was: 2:4215 or .07117+ per cent or approximately .071 per cent.

Bilateral abdominal incision was made in each of the three cases. In two cases, localization of pain was in the epigastrium, in one case with diffuse peritonitis, abdominal pain and tenderness were generalized.

Today localization of the cecum can be determined preoperatively by a flat plate of the abdomen.

SUMMARY

1. The classification of left-sided appendices is: (a) complete or incomplete visceral transposition; (b) non-rotation of colon; (c) imperfect fixation of the cecum and ascending colon; (4) excessively long appendix with the tip to the left of the mid-sagittal line.

2. The incidence of left-sided appendicitis is approximately .071 per cent.

3. Three cases of left-sided appendicitis with appendectomy are reported.

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TUBERCULOSIS OF THE MAJOR BRONCHI

A REVIEW

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INTRODUCTION

The pathologic tuberculous lesions of the major bronchi were described by Eppinger sixty years ago, but little pathologic and practically no clinical attention were paid to them in the ensuing years. After the World War, isolated case reports began appearing in the current literature and the 1920-1930 period saw several cases which were described and diagnosed clinically. Until 1930, however, relatively little was known about the disease. In 1931 Clerf pointed out the value of bronchoscopy in lesions of this nature and this was reemphasized by Sampson in 1936.

With the advent of bronchoscopy into this field, knowledge concerning the nature of the process began to grow. The wealth of material now available has grown mostly in the last five years. Now that our knowledge has reached a relatively ad-

vanced state, it seems advisable to pause and take stock.

DEFINITION

Tuberculosis of the major bronchi, or tuberculous tracheobronchitis, is a complication of pulmonary tuberculosis. The term signifies a specific inflammation of the trachea and major bronchi which involves one or more layers of the wall and includes all types of lesions caused by the tubercle bacillus. It is distinguished from the term tracheobronchial tuberculosis which signifies tuberculous involvement of the tracheobronchial lymph nodes. By the term major bronchus is meant an air passage containing mucosa, submucosa, muscle, encircling cartilage, elastica, and a definite adventitia.

INCIDENCE

The reported incidences of tuberculous tracheobronchitis vary greatly. Flance[8] found it in only 3.1 per cent of patients dying from pulmonary tuberculosis, whereas Bugher, Littig and Culp[3] found an incidence of 41 per cent in a similar series. Other reports are more in agreement. Ophuls found a 10 per cent incidence in 3,000 autopsies. Sampson[19] found a 11 per cent incidence in routine bronchoscopy of tuberculous patients and McIndoe, Steele, Sampson, Anderson and Leslie[13] found the same per cent in 272 patients bronchoscoped. Somewhat higher figures have been found by Myerson[15] and Hawkins.[10] The concensus now is that 10 to 15 per cent of patients with pulmonary tuberculosis have tuberculous tracheobronchitis. It is more frequently found in cases of pulmonary tuberculosis of long standing and in far advanced cases. The complication affects females more than males (probably because the female is more reluctant to bring up the bronchial secretions) and is found in the younger age groups, corresponding with pulmonary tuberculosis.

PATHOGENESIS

Spread to the major bronchi may take place by contiguity, implantation, continuity, or by the blood stream.

The mucous glands of the bronchi are between the cartilage and lumen and separated from the adventitia by muscle. However, accessory lobules penetrate the firm muscle barrier and lie in the adventitia where they are in close proximity to lymph and venous channels draining the adjacent lung parenchyma. Infectious material from tuberculous pneumonia of the peribronchial alveoli, tuberculous lymphangitis and phlebitis, and diseased lymph nodes, therefore, may invade these lobules and spread through the muscle barrier to the mucosa. This is known as spread by contiguity, as advanced by Reichle and Frost.[23] This concept is disputed by Bugher, Littig, and Culp[3] and Cohen and Wessler.[5]

Spread by implantation results from massive eruption of caseous material into the secondary bronchi of the lower lobe. This leads to a spreading infection which soon invades the major bronchi. Bugher, Littig and Culp, and Cohen and Wessler believe that infection usually results from contact with bacteria-laden sputum, the bacilli gaining entrance through the gland ducts or abraded areas.

Parenchymatous disease involves the smaller bronchi. From here tuberculous granulation tissue and caseation may creep by continuity along the submucosa to the major bronchi. All the bronchial structures are destroyed except the mucosa which is left intact.

Hematogenous spread to the bronchi is very rare. Terminal, or agonal, spread just before death may at times occur.

Ornstein and Epstein,[16] while admitting the above, state that tuberculous tracheobronchitis may be a primary lesion without lung involvement or with secondary lung involvement. This is not the generally held opinion.

PATHOLOGY

The fundamental pathology is the same as tuberculous lesions elsewhere in the body. Epithelioid and giant cells, caseous necrosis, granulomatous proliferation, and fibrous tissue are always present. The process, grossly, may be ulcerative, granu-

lomatous, caseous, fibrous, or a combination of these.

Ulcers begin, usually, as a tubercle beneath the mucosa, though they may be the result of direct invasion from the lumen. The infiltration causes interference with blood supply. Caseation of the tubercle then occurs and the mass of necrotic tissue is sloughed out, leaving an ulcer. The surface of the ulcer is irregular, thickened, and may be undermined. A white, homogenous film usually covers the surface; when this is removed a base of granulomatous tissue is found. If the ulcer does not heal, the granulation tissue proliferates to form a tuberculoma which may extend along the bronchi or heal by fibrosis. The granulation tissue looks like any other grossly, but the presence of epithelioid and giant cells may be demonstrated microscopically. If extension does occur, it is always toward the larynx.

Ulcero-granulomatous lesions are representative of the active stage of the disease. They are found most frequently in both bronchial branches and the main bronchus. In the trachea and main bronchi, the lesions are usually on the posterior wall, probably because of the supine position of the tuberculous patient.

Caseous tuberculosis involves all coats. The mucosa and submucosa undergo caseous degeneration and the inflammatory exudate separates the perichondrium from the cartilages. Necrosis of the cartilages results in collapse of the bronchus to an antero-posterior slit with consequent complete or partial obliteration of the lumen. When this last stage is reached the lesion is known as bronchitis obliterans.

Fibrous tuberculosis is the end stage of all forms. Healing is always with excessive scar tissue, the amount varying with the extent of the lesion. The bronchus, by this process, is transformed to a rigid, narrowed tube. Stenosis of varying proportions is the inevitable result and the lesion is then known as ulcerostenotic. Severe stenosis is especially seen following caseous tuberculosis. The collapse of the bronchus seen in this form, with further scar constriction, results in a high grade stenosis. Distal to the stenosis there is an accumulation of infected secretions with subsequent bronchiectasis. Complete stenosis leads to atelectasis. Ball valve stenosis may result either in atelectasis or emphysema.

Lesions are classified, by Eloesser,[6] according to their location, as intramural, mural, and extramural. The intramural lesion is rare; it is the result of a suppurating lymph node breaking through the mucosa and discharging its contents into the lumen. Mural lesions, the most frequent, consist of ulcers, granulomata, infiltrations, and fibrosis. Extramural lesions are the result of pressure by enlarged lymph nodes and peribronchial sclerosis (usually nontuberculous).

Laryngeal tuberculosis, though frequently found with tuberculous tracheobronchitis, bears no causal relationship to it.

SYMPTOMS

The characteristic symptoms are those which result from a decrease in caliber of the trachea and bronchi; the lesions act the same as foreign bodies. The earliest and most constant symptom is a constant wheezing, snoring, or gurgling noise made with each inspiration and is very noticeable by the patient. A sense of oppression under the sternum is felt by some. Dyspnea is present to a varying degree and may be quite severe. It often takes the form of asthmatic attacks; however, the vital capacity is not lowered. Differentiation from asthma is made by the fact that the difficulty is during inspiration, whereas in asthma it is expiratory, and the unchanged vital capacity in tuberculous tracheobronchitis. Cyanosis usually accompanies the dyspneic attacks; it may be transient or permanent.

Paroxysms of coughing, especially when the posture is changed, is a characteristic symptom. Hemoptysis, seldom alarming, may result. Coughing raises an exceptionally thick, tenacious, rubbery sputum which is hard to expectorate. When, after a bout of coughing, a mucous plug is raised, the patient is immediately relieved.

SIGNS

The oral wheeze, so distressing to the patient, may be heard several feet away. Palpable, localized rhonchi, which are not affected by coughing or change of posture, are a frequent accompaniment. Fever, which may be absent, is usually of a low grade type. Sudden elevation to 101-102° usually indicates a plugged bronchus. McIndoe, Steele, and Sampson[13] found some or all of the above symptoms and signs in one-third of their tuberculous patients. Of these, one-third had definite tracheobronchial involvement.

LABORATORY

The sputum is nearly always positive, even though the lung lesions are minimal. X-ray may be of definite value. Atelectasis, either lobar or lobular, is found whenever a bronchus is completely occluded. A ball-valve occlusion may result in emphysema or, if a cavity is present, a "tennis ball" cavity in which air is under positive pressure. Spontaneous, intermittent atelectasis is particularly suggestive; it is the result of edematous or caseous plugging of a bronchus and subsequent re-establishment of the lumen. If the patient is under collapse therapy a sudden decrease in the size of the lung (atelectasis) may appear. The x-ray plate does not show as much parenchymal involvement as one would expect from the clinical picture. Occasionally the bronchial changes are visible. Oechsli's sign is particularly important. This is a thickening along the bronchovascular trunk leading from the parenchymal lesion to the root of the lung with accentuated visibility of the bronchial lumen; it is probably due to submucosal infiltration.

DIAGNOSIS

The diagnosis is suspected in any patient with tuberculosis exhibiting any of the symptoms and signs suggestive of tuberculous tracheobronchitis. A persistently positive, more profuse sputum containing many plugs and casts, with minimal pulmonary involvement is very suggestive. A partially obstructed cavity with a fluid level may exhibit a rapid enlargement which cannot be explained on a basis of tissue destruction; this is indicative of a ball-valve bronchial obstruction. Pulmonary tuberculosis very rarely produces atelectasis or emphysema; these findings, therefore, should lead to a suspicion of tuberculous tracheobronchitis. Serial plates are necessary to show the changes and to pick up a transient atelectasis. Although a bronchogram with lipiodol will reveal a stenosed bronchus, it is felt that it is a dangerous procedure because of the iodine content of the lipiodol.

Positive diagnosis is made by bronchoscopy, which should be done in all patients exhibiting any of the findings pointing to a suspicion of tuberculous tracheobronchitis.

DIFFERENTIAL DIAGNOSIS

Tuberculous tracheobronchitis must be differentiated from non-specific ulcers, erosion from lymph nodes, syphilis, neoplasms, and rhinoscleroma. Differentiation is best made by bronchoscopy.

The tuberculous lesion, bronchoscopically, is a chronic thickening and congestion of the mucosa. Jackson describes a peculiar cyanotic tint of the surface. Petechial hemorrhages may be present. Ulceration is common.

Erosion of a lymph node through a bronchus is diagnosed by the sudden, severe, bout of coughing followed by expectoration of the node. Bronchoscopy reveals a perforated ulcer of the bronchus.

Syphilitic involvement is usually a third stage gumma. Obstruction is not the rule, nor is dyspnea usually seen. Bronchoscopically, a constructive process is seen, although ulceration may be present. Bleeding is not the rule. Biopsy proves the diagnosis.

Bronchiogenic carcinoma is seen after middle age. Hemoptysis is much more frequent. The lesion is fungating and obstructive and bleeds easily. A malignant ulcer, if present, has rolled, indurated edges. Such lesions should always be biopsied.

Rhinoscleroma is also seen after forty. The nodules characteristic of this disease coalesce to form an infiltrative mass of a chronic inflammatory character. The lesion

is hard, dark brown, or red shade, and does not ulcerate. Biopsy should be done.

COMPLICATIONS

Complications are atelectasis, bronchiectasis, stenosis, and infection of uninvolved parenchyma. Atelectasis is a frequent finding and may be regarded as part of the symptom complex. It is, however, an undesirable feature because infected secretions accumulate behind the stenosis where they may involve healthy tissues and produce an exacerbation of symptoms. Factors conducive to the production of atelectasis are bed rest, shallow breathing, shortening and kinking of the bronchi by scar tissue, bronchoscopic examination, and phrenicectomy.

Bronchiectasis is the result of the damming back of secretions behind a stenosis with resultant abscess cavities, inflammation, congestion, and swelling. It is a frequent finding; Cohen and Higgins[4] found bronchiectasis in 17 out of 19 patients with tuberculous tracheobronchitis. It usually comes on several months after bronchial involvement. Unsuspected fever associated with a sudden rise of temperature is indicative of retention of secretion. Diagnosis is by bronchoscopy.

Stenosis of varying degree is an inevitable complication. When this is severe, portions of a lung, or even a whole lung becomes functionless. High grade stenosis of the trachea causes death by suffocation.

It should be emphasized that tuberculous tracheobronchitis is often responsible for involvement of the contralateral, uninvolved lung. Sudden, unexpected appearance of lesions on the opposite side should lead to a suspicion of extension from a bronchial lesion.

PROGNOSIS

The prognosis of tuberculous tracheobronchitis is very poor. Sampson, Barnwell, Littig and Bugher[20] report a 52.9 per cent mortality. Other series,[21] show practically the same percentage deaths. No patient ever fully recovers, although he may live for several years. Since tuberculous tracheobronchitis is seen mostly in cases of far advanced tuberculosis, it is not surprising that the mortality is high. Death usually results from suffocation or the parenchymal process.

Warren, Wadsworth, Hammond and Tuttle[24] are not as pessimistic. They state that in 54 patients treated (by Kernan electrodes) 34 (59 per cent) were healed, 10 progressed or did not improve, and nine are still under treatment. These cases, however, were not advanced. Simple ulcers certainly carry a better prognosis.

TREATMENT

Treatment is very unsatisfactory and there is no unanimity of opinion concerning procedure. Palliative treatment consists of cough mixtures, croup tents, bed rest, and antispasmodics. None are of much value except bed rest. Aspiration of pus and secretions through the bronchoscope affords relief. Sampson[22] advocates the application of epinephrine and cocaine to the edematous areas. These measures afford temporary relief only. Elimination of allergens and administration of tuberculin, as advocated by some, is useless and may be dangerous.

Deep x-ray therapy was advocated by Epstein and Ornstein[7] in 1936 but trial proved its worthlessness and it is not used at present. Ultraviolet radiation is now placed in the same category.

Epstein and Ornstein,[7] and Myerson[15] believe the simple ulcer should be left alone and allowed to heal spontaneously; only the indolent ulcers should be treated. Kernan,[12] however, advises treatment of simple ulcers because he believes they will then heal with less scar.

Concerning collapse therapy there is a diversity of opinion. Barnwell, Littig and Culp, and Epstein and Ornstein believe that it is indicated by atelectasis, senosis, and the pulmonary lesion. Ornstein and Epstein[16] state that pneumothorax is desirable because it reduces air flow and puts the bronchus at rest, prevents spread, diminishes pulmonary vascularity, and relieves wheezing. They state that three out of eight patients had symptomatic relief

following pneumothorax. Sampson[21] states that the results of collapse are the same as no treatment at all and therefore advocates it only in so far as the pulmonary lesion is concerned. However, Jenks[11] states that collapse is contraindicated and should not be done until after the lesions have healed.

Cauterization with the electric cautery is advocated for tuberculomatous lesions by many.[7,15,19] Kernan,[12] however, points out that, although healing is rapid, there is much resultant scar tissue with danger of stenosis, and reports a case of fatal hemorrhage following erosion of a large vessel by the cautery. Silver nitrate is also used. Goldberg[9] cautions that it should not be used in a strength over 5 per cent because of the greater formation of scar tissue. Kernan suggests the use of the mercury vapor lamp in cauterization, stating that less scar is produced and the method is safe. It should be applied two to three minutes every two weeks. When scar has formed, he recommends copper ionization which softens the scar and permits dilatation. A current of three milliamperes is allowed to act two or three minutes once a week.

Dilatation of stenosed bronchi should be carried out with great caution, if at all. Benedict[2] believes it should always be tried, and points to success in three cases. Sampson agrees with him. On the other hand the dangers of ulceration and perforation are great. Myerson[15] believes that dilatation is impossible because of the extent and firmness of tuberculous stenosis, and therefore should never be attempted. Dilatation and shrinkage with aspiration of retained secretions is of great temporary value.

Kernan,[12] and Cohen and Wessler[5] believe that complete stricture of a bronchus might be desirable if the distal tissues are uninfected; it is the small openings in the strictures present which permit infection of the distal parenchyma without permitting useful function. To date no one has attempted this suggested form of therapy.

Sampson[19] has suggested lobectomy, pneumonectomy, or external drainage in selected cases. To date these measures have not been tried, to my knowledge.

SUMMARY OF THE ROLE OF BRONCHOSCOPY

The bronchoscope is responsible, more than any other thing, for the growth of knowledge of tuberculous tracheobronchitis. Prior to 1935 it was generally thought that bronchoscopy was contraindicated in tuberculous patients. Sampson,[22] in 1936, showed that there is relatively little danger and that bronchoscopy is indicated when tuberculous tracheobronchitis is suspected. By use of the bronchoscope the condition may be accurately diagnosed and treated. Although there is little of permanent positive value the bronchoscopist can do, less can be accomplished by other means.

Bronchoscopy is indicated in any tuberculous patient in whom tuberculous tracheobronchitis is suspected. It is contraindicated in acute laryngeal disease, when there has been a recent severe hemoptysis, and when the patient is acutely ill. Jenks[11] points out that the cough reflex is abolished in bronchoscopy and infectious material is loosened from the walls by the instrument, with the consequent danger of aspirating the material and spreading the disease. Although this is theoretically so, the danger of spread by this means is not great. Warren, Wadsworth, Hammond, and Tuttle[24] believed that bronchoscopy was responsible for spread in two of 57 cases studied.

Bronchoscopy is also of value before thoracoplasty and other major collapse procedures, and it is the concensus that it should be done routinely in these cases. Frequently the bronchoscopist will find tuberculous lesions of the bronchi on the side opposite that of contemplated surgery. Obviously, permanent collapse of one lung with the other threatened with a stenosing bronchitis is definitely contraindicated.

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SOME RECENT ADVANCES IN GASTROINTESTINAL PHYSIOLOGY

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NEW ORLEANS

INTRODUCTION

Although speculation as to the mechanics of the gastrointestinal tract has gone on for centuries, significant advances date back to 1825. At this time, William Beaumont began his classic experiments on gastric juice and digestion. Alexis St. Martin, a Canadian of French descent, was wounded by the discharge of a musket, and went to Beaumont for treatment. After a rather hectic and protracted period, St. Martin's health returned, but he was left with a permanent gastrostomy. Beaumont, realizing the experimental possibilities, worked laboriously for many years, and in 1833 reported the results of his painstaking research in a book entitled "Experiments and Observations on the Gastric Juice, and the Physiology of Digestion." This work marks the beginning of accurate knowledge of gastrointestinal physiology, for, in it, Beaumont disproved many of the absurdities that were regarded as scientific facts at the time.

If one reads a text or work written in the first part of the nineteenth century, he will be amazed at the remarkable advances that have been made in the last 100 years. For instance: Spallanzani, in 1793, after numerous experiments, declared the gastric juice to be entirely neutral and inert. It was commonly believed that hunger was due to friction of the inner walls of the empty stomach, to the mechanical action of the diaphragm, or to dilatation of the gastric vessels. Montegre contended that the gastric juice was nothing but saliva, that its principal use was to dilute the food, and that the stomach possessed a peculiar sensibility in that it had absorbent vessels which could take up certain parts of the food and reject others. Jackson believed that digestion was performed by submitting food to the action of different fluids, each of which had "solvent powers for different principles."

Obviously, noteworthy advances have been made in the last century. Too, much has been done in the last few years to clarify the intricate problem of gastrointestinal physiology. Most marked advances have been made in the fields of motility, secretion, and absorption. To say the least, all

questions are not settled, but significant advances are being made every day.

GENERAL

Crandall et al. claim that the P-P factor (nicotinic acid) is essential for the maintenance of normal gastrointestinal motility. Vitamin B deficient animals display a craving for fat and an aversion to carbohydrate and protein food; this aversion may be the result of disturbed gastrointestinal physiology or it may be due to a disturbance in the metabolism of these two foodstuffs.

Recently, by means of a special resistance thermometer, the temperature of the normal gastrointestinal tract was determined. For males, it varies between a maximum of 101.8°F. and a minimum of 97.8°F., with an average of 99.2°F.; for females, it varies between 97.5°F. and 102.2°F., with an average of 99.4°F. It was found that drinking 250 c. c. of water at 40°F. caused a drop in the gastrointestinal temperature of 30-60°F. with advance of the motor end point; the average emptying of the stomach was delayed 30 to 45 minutes and occasionally longer. Hot coffee causes an increase in temperature of 4-17°F. with a recovery time of 45 minutes; however, there is no appreciable change in gastric emptying time. Application of ice bags to the abdomen for an hour causes a decrease of only 1°F. in intragastric and intraduodenal temperature. Thus, if application of an ice bag is of any benefit in cases of gastric and duodenal hemorrhage, it is not due to lowering of the gastrointestinal temperature.

PEPTIC ULCER

More evidence that hyperacidity is unimportant in the etiology of peptic ulcer has been advanced recently. Dogs were sham fed 10 to 12 hours a day without obtaining the least evidence of chronic ulcers; too, there is no correlation between acidity and degree of distress or acidity and recurrences.

Martin and Schnedorf have made an extensive investigation of the claims that lesions of the hypothalamus produce peptic ulcers. By placing localized lesions in the hypothalamus of seven monkeys and 40 cats, they were unable to effect a change in gastric secretion or motility, nor did autopsy show pathologic lesions in the gastrointestinal tract.

Mann and Bollman report that the oral, rectal, and parenteral administration of cinchophen caused the uniform development of peptic ulcers in animals. Pectin has a prophylactic and curative action for these ulcers, for when it was given with cinchophen, the incidence of ulcer formation was reduced to 11 per cent.

Noting that peptic ulcers occur only infrequently in pregnancy (and, if present, show a tendency to remission), Sandweiss et al. studied the effect of sex hormones on the incidence of ulcers in dogs with a Mann-Williamson operation. They found that pregnancy-urine extract (antuitrin-S) definitely decreased the incidence of ulcers in such animals. There is as yet no feasible explanation for the action of this extract.

Although no evidence of beneficial effect of ascorbic acid was noted when administered to ulcer patients, Chamberlin and Perkin found the blood and urine of ulcer patients to contain less ascorbic acid than normal.

Recently, two additional theories of the etiology of peptic ulcer have been advanced. Both contend that a vasoconstrictor acts on the stomach, devitalizing the tissues and rendering them susceptible to the digestive action of hydrochloric acid and enzymes. Necheles believes the etiologic factor to be acetylcholine; Babkin believes it to be histamine. Other investigators have shown that 37 per cent of dogs receiving repeated injections of pitressin develop ulcers; apparently they result from vascular interference with local nutrition in the gastric mucosa.

INTESTINAL OBSTRUCTION

Evidence has been presented by Montgomery which shows that obstruction of the intestine does not increase secretion, thereby producing a vicious cycle. Wangensteen et al. have strikingly demonstrated the injurious effects of distention in intestinal obstruction. These workers ligated the esophagus and terminal ileum of dogs and

found that they were able to live 36 to 57 days. Autopsy showed the gut to be collapsed with complete resorption of digestive juices. Such a procedure prevented aerophagia with consequent distention, increased intraluminal pressure, and decreased viability of the gut. Evidently, the mechanical factor of distention is more important than the "toxic factor" as the cause of death in ileal obstruction. Other evidence has been presented to show that toxemia is less important in intestinal obstruction than demineralization and dehydration.

SALIVARY SECRETION

Gantt has shown that there is a direct linear relationship between the weight of food eaten and the volume of the resulting salivary secretion. The time consumed in eating was found to have no significant effect on the response of the salivary glands.

Langstroth et al., by applying spectroscopic methods to saliva secreted in cats in response to stimulation of the chorda tympani nerve, report that a direct linear relationship exists between the sodium concentration of saliva and the volume-rate of secretion, whereas potassium concentration remains constant and independent of volume rate. At very low rates of secretion, however, the concentration of both sodium and potassium increases, revealing a significant degree of absorption of water at these low rates. The protein concentration of the saliva is also increased with the rate of secretion, but as stimulation continued, its concentration tended to drop as a result of exhaustion of its intracellular precursors.

Kesztyus and Martin found saliva produced by sympathetic stimulation in dogs to be higher in potassium, calcium, organic solids, mucin, and albumin, and lower in sodium and chloride than saliva obtained by chorda stimulation. Apparently chorda stimulation produces saliva in the newborn. This saliva remains constant in composition until the thirtieth to fortieth day when coincident with the appearance of sympathetic innervation, its composition suddenly changes.

Pierce and Gregersen, by comparing the response of dogs with chronic salivary fistulae to continuous intravenous injection of pilocarpine before and after unilateral section of the chorda tympani, found, contrary to their expectations, the response of the denervated gland to be augmented. After two or three weeks, high levels were reached which persisted at least a year. This augmentation was not due to increased blood flow or to accumulation of preformed saliva in the duct passages. The authors do not attribute this to a specific sensitization produced by denervation since acetylcholine gave a greater response on the normal than on the denervated side.

Gibbs and McClanahan report that histamine, in contrast to acetylcholine, produces an irregular secretory response in the salivary gland when injected in minute doses directly into the arterial supply of the gland. When the stimulating effect of histamine was present, it was abolished by atropine and potentiated by eserine. Too, histamine did not potentiate acetylcholine stimulation, but did lengthen the effect of chorda stimulation. Thus histamine may act by permitting a continuation of the process initiated by nerve stimulation; it probably removes a natural "brake" mechanism. If this is true, it may be of significance in understanding paralytic salivary secretion.

Meyer et al. have reported that the ptyalin content of saliva is greatly reduced in old age. A protein formed by the salivary gland supposedly behaves as thrombokinase and is thus responsible for the blood-coagulating action of saliva. It has been shown that intravenous injection of saline produces thirst and reduces salivary flow.

Ivy et al. have shown that smoking stimulates the flow of saliva reflexly, that is, the smoke irritates the buccal mucosa. The flow of saliva was not stimulated by the nicotine absorbed from two or three cigarets.

THE STOMACH

Bussabarger, Freeman, and Ivy found that removing the stomach from growing puppies caused faulty ossification of bones, even though the diet was adequate. The result was a homogeneous osteoporosis

which was often so severe that spontaneous fractures occurred. This osteoporosis was analogous to that observed in celiac disease without rickets. Apparently this deficient ossification was due to a combination of several factors, among which are the following: (1) there was an absence of hydrochloric acid, which normally renders the calcium salts more soluble and maintains an acid state in the intestine; (2) there was an increase in intestinal transport rate as the stomach no longer served as a reservoir; (3) calcium retention was not favored by postcibal acidosis. It has been shown by other workers that gastrectomies in growing pigs resulted in anemia, skin and spinal cord changes, osteoporosis, and extreme emaciation.

GASTRIC SECRETION

Pickett and Van Liere point out that appreciable degrees of anoxia distinctly diminish the volume and acidity of gastric juice, probably by depressing the activity of the secretory cells of the stomach. That the vagus was concerned was shown by the fact that the group of dogs with Heidenhain pouches was affected by less severe degrees of anoxia than the Pavlov group. Alvarez and Vanzant have shown that a fall of hemoglobin, such as is caused by anemia, to 75 per cent, is followed by a decrease in gastric acidity. Thus both anoxic and anemic types of anoxia produce decreases in gastric acidity.

Ivy et al. claim that smoking of an ordinary number of cigarets increased the acid output of the fasting stomach in only two of sixty human subjects, and the increase was so slight as to be of doubtful practical significance. They found depression of gastric acidity to be much more common. Thus they conclude that smoking is not conducive to peptic ulceration, nor does it aggravate its symptoms.

It is a well established fact that gastric secretion and motility are inhibited by the ingestion of fat. Farrall and Ivy proved this inhibition to be a humoral one, as oral administration of fat was found to inhibit motor activity of the transplanted and denervated gastric pouch. Quigley and Ivy subsequently showed that sugars likewise inhibit gastric motility by a humoral mechanism. It has been demonstrated that the humoral agent is not fat or one of its products of digestion, nor is it bile, secretin, or cholecystokinin. Lim and his coworkers have proved this humoral agent to be a specific duodenal cholane and have successfully prepared extracts of duodenal mucosa which inhibited gastric secretion and mobility. This active principle was given the name enterogastrone.

In view of the wide variations between the ratio of secretory to motor inhibitions of various preparations of enterogastrone, it has been suggested that this substance consists of two principles—one which inhibits secretion and one which depresses motility.

Sandweiss et al. recently reported that extracts of pregnancy urine (antuitrin-S) are potent in preventing the development of jejunal ulcers in dogs subjected to the Mann-Williamson operation. Both Necheles and Sandweiss have prepared extracts from normal female urine which is potent in inhibiting gastric secretion of dogs; this definitely excludes prolan as the active constituent as it originates in the placenta. Similar extracts of male urine are likewise potent in decreasing gastric secretion; however, no inhibition of gastric motility even with large doses could be observed by Ivy et al. Nechele and his coworkers claim that their urine extracts abolish or inhibit both gastric secretion and gastric motility in doses of 1 to 4 mg. As both the chemical and biologic behavior of this substance in the urine resembles the duodenal preparation of enterogastrone, it is likely that enterogastrone is excreted through the urine.

Quigley et al. recently investigated the claims that secretory activity of the gastric glands is accompanied by changes in electrical potentials of the gastric mucosa. They recorded the difference in potential between the inner surface of the stomach or pouch and the skin in dogs with a Pavlov pouch, but they could detect no changes accompanying either gastric secretion or motor activity. Milk, dextrose, and alcohol

lowered the potential of the pouch when they were introduced into the main stomach, but not when given intravenously. Emulsified fat has no such action; thus enterogastrone is eliminated as the agent responsible for the depression of potentials. The unknown activity on which the potential depends is abolished by the intravenous injection of sodium cyanide.

Some investigators still claim that the hormone, gastrin, is identical with histamine. However, it is likely that histamine is not gastrin, as atropine will abolish the gastric secretory response of dogs to a meal but will not abolish histamine secretion. There is apparently no increase in histamine in dog's blood during digestion of a meal. However, histamine is found in gastric juice in higher concentration than in the blood after sham feeding and histamine injections. The concentration of histamine increased with the rate of secretion of juice. Blood histamine is considered to be found in the formed elements and not free in the plasma; the blood histamine concentration remains unchanged on passing through the stomach. Thus, histamine is apparently formed in the gastric mucosa and secreted by the parietal cells. Some believe that histamine mediates the secretion of the parietal cells; the acetylcholine liberated by the vagus nerve liberates histamine which stimulates the parietal cell. Thus histamine is considered to be necessary in the normal secretory process of the parietal cell, but it is separate and distinct from the hormone, gastrin. It is claimed that histamine-free extracts of the pyloric mucosa, given intravenously, stimulate gastric secretion, and are not affected by atropine. The question as to whether or not histamine stimulates pepsin secretion is still unsettled.

Wilhelmj et al. have shown that the presence of high acidity in the stomach inhibits the gastric phase of secretion, and that high acidity in the intestine inhibits the intestinal phase of secretion.

Recent workers have shown that small doses of acetyl-B-choline produce a copious flow of highly acid gastric juice; large doses, however, have a reverse effect. Both of these effects are abolished by atropine. This reversal of effect with dosage probably explains the contradictory reports in the literature as to the effect of choline derivatives on gastric secretion.

Studies on patients with achylia gastrica showed that they do not show a volume response to injections of histamine. Chemical studies proved that the parietal cells had made no contribution to the secretion.

It has been shown that the "intrinsic" factor of Castle is not elaborated solely by the gastric mucosa, for a potent anti-pernicious anemia preparation can be made from desiccated duodenal mucosa of hogs. It is claimed that normal gastric juice, in contrast to gastric juice obtained from patients with pernicious anemia, possesses proteolytic activity not due to pepsin, but, to the "intrinsic" factor. This is not a settled question by any means. Since the "intrinsic" factor is inactive at acidities below pH 2.5, Castle et al. suggest that this factor operates parenterally and not in the gastrointestinal tract. They also claim that the incubation of the "intrinsic" and "extrinsic" factors does not result in the formation of the thermostable principle in the liver.

Alcohol, given intravenously, increases the volume of gastric juice and lowers the total and free acid, according to Lolli, but there was no augmentation of mucus as there is following oral administration. Spies et al. point out that chronic alcoholism reduces gastric acidity and volume but not peptic activity.

Morrison believes that neutral red is eliminated from the gastric mucosa exclusively through the parietal cells; thus a test of secretory activity of these cells is afforded.

It has been pointed out that normal gastric juice has no effect on gastric secretion when injected into dogs; however, juice from subjects with pernicious anemia or cancer produced periods of achlorhydria. The active principle proved to be heat labile.

The effect of acids and alkalis on gastric emptying time has engaged the attention of

physiologists and clinicians for many years. Kussmaul and Hirsch are usually regarded as the pioneer workers in this field. Most investigators agree that gastric motility is inhibited when strong acids are added to the gastric contents, whereas the effect of weak acids is indeterminate. Too, most workers agree that alkali in small doses increases the number and depth of gastric peristaltic waves; thus average doses of sodium bicarbonate generally hasten stomach emptying. There is considerable evidence, however, that large doses of alkali inhibit gastric motility.

The acid-control theory of the pylorus suggested by Cannon in 1898 is no longer tenable. It is known, for example, that the achlorhydric stomach empties faster than the normal stomach; too, alkalis in moderate doses hasten gastric emptying.

Some evidence has been presented that the mechanisms controlling secretory and motor function of the normal stomach, in response to a meal, must be identical. This is certainly not true of achlorhydrics, whose stomachs often empty even more rapidly than normal, or in ulcer patients, who may have marked retention in stomachs with a relatively high acidity, even in the absence of organic obstruction. However, if the mechanisms controlling secretory and motor functions are identical in the normal stomach, it is logical that when acid is added to the gastric contents part of the need for secretory function is removed, so that less secretory work is done, and concomitantly, the motor function may be somewhat decreased. Conversely, if alkali be added, there is a stimulus for added secretory function—and thus there is augmentation of motor function. Some workers feel that there is no definite correlation between gastric acidity and motility, however.

Numerous investigators have shown that colloidal alluminum hydroxide gel exhibits considerable antacid action not only *in vitro* but *in vivo*. There is considerable disagreement, however, as to whether or not colloidal aluminum hydroxide exerts a stimulating effect on gastric secretion. Ivy et al. noted slightly higher acid values for gastric contents after a plain test meal in dogs treated with aluminum hydroxide; Emery and Rutherford found an increase in the secretory response to histamine after 10 weeks' treatment with aluminum hydroxide. Einsil et al. were unable to duplicate the latter findings, however.

Komarov and Kreuger claim that colloidal aluminum hydroxide actually inhibits gastric secretion. Using dogs with Pavlov pouches, they found that the introduction of aluminum hydroxide into the main stomach often inhibited the gastric secretion in the isolated pouch. This inhibitory effect was most marked on the peptic power of the gastric secretion.

Coffey et al. report that hypnotic and anesthetic doses of barbiturates seem to decrease significantly gastric and pancreatic secretion. The hyposecretion of gastric juice following barbiturates may explain, at least in part, the benefit of these drugs as adjuncts in the therapy of peptic ulcer. Rather significant amounts of barbiturates were excreted in the gastric juice; however, it is likely that gastric lavage, unless repeated frequently, is not warranted in poisoning from large amounts of barbiturates.

GASTRIC MOTILITY

By means of three tandem balloons located in the pyloric antrum, the pyloric sphincter, and the duodenal bulb, Meschan and Quigley studied the gastric outlet. They found that these three regions operate as one functional unit; waves which originate in the stomach pass successively over all three structures. The sphincter operates to prevent regurgitation to a much greater extent than to regulate evacuation, for it tends to be passively relaxed most of the time, contracting only when a peristaltic wave hits it. This contraction persists while the wave passes over the duodenal bulb. Vagotomy does not disturb this complex, coordinated mechanism. There was no indication of antagonistic effects on the viscus and sphincter as is so generally presumed to occur. Fats, fatty acids, dextrose, soaps, and dilute mineral acids inhibit motility in the pyloric region. Bilateral vago-

tomies markedly decrease or abolish these inhibitory effects, indicating that they operate mainly through the enterogastric reflex.

By employing a differential manometer, Thomas was able to determine the differences of pressure between the duodenal and gastric sides of the pyloric sphincter in fistula dogs. When the stomach contained food, the intragastric pressure was generally higher than that in the duodenal bulb, even during periods of relative inactivity. During evacuation of solid foods, cyclic changes in pressure were observed which consisted of first an increase in intragastric pressure over intraduodenal, followed by a return of the intragastric pressure to that of the duodenum, and finally a rise of the intraduodenal over the intragastric pressure. Such a cycle probably represents the passage of a peristaltic wave. Frequently when liquids were evacuated from the stomach, incomplete cycles were noted. Typically, there was a lack of the initial augmented intragastric pressure. Direct proof that the pyloric sphincter plays only a secondary role in the regulation of normal gastric evacuation has been advanced by Crider and Thomas. They could detect no change in the emptying time of 5 per cent glucose, 0.05 N hydrochloric acid, olive oil, 10 per cent alcohol, or isotonic saline when the pyloric sphincter was kept open continuously by means of a special perforated tube.

Thomas and Crider observed that commercial peptones, intestinal contents of dogs during protein digestion, proteases, certain free amino acids (especially the monoaminocarboxylic acids), or enzyme or acid digests of casein, when introduced into the lower duodenum of dogs, produce inhibition of pyloric antral motility with a corresponding delay in gastric evacuation. As the latent period was frequently about 15 seconds and vagotomy abolished this inhibition, it is likely that the vagi constitute the afferent pathway. Some of the individual amino acids appear to have no inhibitory effect; in general, the latent periods for inhibition following the injection of proteose preparations were slightly longer than those of other substances. Acidity was found to be a major factor in gastric inhibition only at pH's less than 3; thus, the acidity of chyme is of much less importance in the normal regulation of gastric evacuation than are the digestion products of food. Other investigators have shown that fat and carbohydrate in the intestine inhibit gastric peristalsis and delay gastric emptying. In order of inhibitory potency, fats come first, products of protein digestion second, and carbohydrates third. Cannon has pointed out that the gastric emptying times for the three types of food decrease in the same order.

Gastric inhibition is obtained by injection of the duodenal hormone, enterogastrone. Occasionally refractoriness is developed to repeated injections; more than likely this is due to impurities in the extract for continued response to the hormone elaborated physiologically has repeatedly been noted.

Brucke and Stern have investigated the innervation of the cardiac sphincter in cats, attempting to locate the adrenergic and cholinergic fibers and study their functions. They found that bilateral vagotomy produced a cardiospasm which was abolished by atropine and epinephrine, but exaggerated by eserine, acetylcholine, and pilocarpine. Too, stimulation of the peripheral end of the vagus produced relaxation of the sphincter. Thus the vagus was inhibitory to the sphincter; this was an effect of adrenergic fibers for the action was abolished by ergotamine and potentiated by cocaine. Similarly, stimulation of the splanchnics produced cardiospasm; this was due to cholinergic fibers as the action was potentiated by eserine and abolished by atropine. The vagus sometimes relaxes and sometimes contracts the stomach according to the tonus level.

It is important to classify autonomic nerves and interpret their function on the basis of their chemical mediator rather than on their anatomic origin. Proof of this is in the work of Gayet et al. who reported that stimulation of the splanchnics in vagotomized, atropinized, and eserinized

dogs with the adrenals removed liberated acetylcholine into the venous blood from the stomach regularly, from the small intestine, irregularly, and from the pancreas, inappreciably. Thus autonomic nerves are frequently mixed ones.

It has been shown that section of the splanchnics increases the motility of the empty stomach both as to duration of periods of activity and amplitude of contractions.

A new type of physiologic mechanism for accomplishing gastric and intestinal motor inhibition has been reported by Youmans and Meek. They found that mechanical stimulation of the rectum and anus in dogs inhibited gastric and jejunal motility and tone after vagotomy and removal of the adrenal medulla. This effect was abolished by chain ganglionectomy and splanchnic section; apparently, mechanical stimulation of the colon liberates a humoral inhibitor from the sympathetic nerve endings of the colon.

It has been shown that insulin has a stimulatory effect on gastric motility; this effect is mediated by the vagi. However, insulin probably has a peripheral inhibitory action as manifested by the fact that vagotomy not only abolished the excitation, but converted it to inhibition.

Stretching the rectum of a dog with pressure of 20 to 48 mm. of water inhibits the motility and tone of the empty stomach. However, recovery usually takes place while the pressure is maintained. Undiluted gallbladder bile placed in the rectum inhibits hunger contractions yet normal saline and 10 per cent glucose do not. Uspensky found that the application of heat and cold to the abdomen has variable effects on human gastric tone and mobility, depending partly on the previous state of the stomach.

Scott et al. contend that there is a lack of agreement between hunger sensations and hunger contractions. In man, the administration of alcohol inhibits hunger contractions yet hunger sensations are more intense. This group also found no causal relationship between blood sugar values and normal hunger periods; apparently, the blood sugar level remains constant during periods of gastric activity and rest. However, Blotner finds insulin to be of value in malnutrition due to nervous dyspepsia; he believes insulin hypoglycemia induces hunger by augmenting hunger contractions.

Quigley et al. found that prolonged administration of large doses of aluminum hydroxide did not significantly alter the histology of the gastric tissue, the motility of the pyloric antrum, sphincter, or duodenal bulb, or the gastric evacuation time.

Ivy and Schnedorf contend that hunger contractions in man cease after the first few puffs of cigaret smoke; often smoking one cigaret inhibits the contractions for 15 to 60 minutes after the cessation of smoking. Tobacco smoke inhibits hunger contractions through a reflex mechanism, the motor side of which is in the vagi; small doses of nicotine do not have this inhibitory action. However, moderate smoking does not delay the emptying of a test meal from the stomach.

DUODENUM

The question of whether or not there is a duodenal hormone involved in carbohydrate metabolism is a much debated one. Reports that the injection or ingestion of certain extracts of the intestinal mucosa or stimulation of the duodenal mucosa with dilute hydrochloric acid causes a reduction of blood sugar concentration have led to a concept of a duodenal hormone which aids in the regulation of carbohydrate metabolism. Conti has reported that the hyperglycemia of diabetics is reduced by oral and duodenal administration of dilute acid. However, in carefully controlled experiments, Ivy, Loew, and Gray failed to yield evidence that acid stimulation of the duodenum reduces the hyperglycemia produced by absorption of glucose, intravenous injections of glucose, adrenalin injections, or removal of the pancreas. Thus, they conclude that acid stimulation of the duodenum has no influence, either humoral or nervous, upon carbohydrate metabolism. This same group has found that pancreatic-tissue free extracts prepared from intesti-

nal mucosa by secretin, insulin, and enterogastrone methods, consistently failed to lower the fasting blood sugar levels of dogs. This evidence makes it extremely doubtful as to the presence of a hormone liberated by the duodenal mucosa which aids in the control of carbohydrate metabolism.

INTESTINAL MOTILITY

Youmans et al. observed that distention of the intestine led to motor inhibition of the gut above and below the site of distention. This inhibition passed chiefly over extrinsic nerve pathways, although some effect could be obtained over the intrinsic nerves when the extrinsic paths were cut.

Reid, Ivy, and Quigley have described spiral movements of the intestine. Such movements are probably produced by the "close spiral" muscles and are often independent of the longitudinal movements produced by the "long spiral" muscle. Weak electric stimulation of the anterior hypothalamus, superchiasmic region, or ventral portion of the supramammillary decussation results in movements of the gut.

Morphine produces a transient increase in both propulsive and non-propulsive motility followed by high tone and complete loss of propulsive motility.

Stimulation of the vagus results in augmented motility of the villi, while stimulating the splanchnics produces inhibition. "Villikinin," the hormone which increases the motility of the villi, is liberated by the introduction into the intestine of dilute hydrochloric acid but not glucose. Such an increased activity of the villi by the presence of hydrochloric acid results in faster absorption of glucose from the duodenum.

Alvarez has shown that rhythmic contractions in the small bowel are constant for any given segment. Thomas and Kuntz, by giving sufficient doses of nicotine to paralyze the synapses in the intrinsic intestinal plexuses, were able to arrest all types of motility which depend on the integrity of the plexuses, yet rhythmic contractions proceed regularly and constantly. They concluded that the rate of rhythmic contractions is probably a function of intestinal musculature. The rate of rhythmic contraction of any given segment of bowel is a function of its distance from the pylorus, the loop at the highest level having the highest rate, that at the lowest level having the lowest rate. The rate is unaffected by fasting, feeding, or sleep, except that in the recently fed animal rhythmic contractions were less commonly observed than in the fasting animal. The rate was unaffected by degenerative vagal or splanchnic section.

A large number of inhibitory reflexes have been described, yet only two motor reflexes are known: the gastro-ileac reflex and the gastro-colic reflex, both of which are concerned with the ingestion of food. It has been known since 1886 that the ingestion of food is followed by increased motility of the ileum. Strikingly enough, there has been no convincing demonstration that the vagi, which are believed to be motor nerves of the small intestine, play any part in mediating motor reflexes. That this activity of the ileum to the ingestion of food is not entirely a psychic one is borne out by two findings: (1) merely presenting the meal to the animal without allowing it to eat does not result in the motor response of the ileum; (2) the motor response is as constant after feeding by a gastric fistula as after normal feeding. Douglas and Mann report that this response occurs constantly after double thoracic vagotomy; this was unexpected but does lend evidence to the independence of the intrinsic enteric mechanism. This does not prove entirely that the vagi do not play a part in mediating the motor response in the normal animal, but, in view of the marked similarity of response and time interval in normal and vagotomized animals, another means of mediation seems likely. As short-circuiting or complete isolation of the bowel decreased or abolished this response, even though the extrinsic nerve supply was intact, it is evident that the continuity of the intestine is necessary for this motor response. These findings also tend to indicate that there is no humoral mechanism involved. These coworkers claim that the type of activity, as well as the degree of activity, is governed

by the ingestion of food. Thus, in the fasting animal, there was little movement except for gentle segmentation, while, in the recently fed animal, there were peristaltic waves, tonus changes, and propulsive forms of segmentation.

There is no question that the thyroid gland exerts some influence on gastrointestinal physiology. All agree that this gland causes increased gastrointestinal motility when it hyperfunctions. Investigators have been trying to determine through what mechanism the abnormally acting thyroid gland exerts its effect upon the digestive tube. Many contend that the increased gastrointestinal motility in hyperthyroidism is produced by direct stimulation of the vagus by the hyperfunctioning gland. Morrison, Feldman, and others have shown that induced hyperthyroidism in the vagotomized dog produces increased gastrointestinal motility; thus the thyroid activity is probably independent of the vagus nerves. It is believed that the heightened basal metabolic activity accounts for this increased motility.

The possible existence of a humoral mechanism for the regulation of intestinal motility has been investigated. By introducing 0.4 per cent hydrochloric acid into the duodenum of a dog, under morphine-ether anesthesia, whose intestine had been completely denervated, it was shown that a flow of pancreatic juice resulted, but no effect on the motility of the intestine was noted. Such evidence is by no means conclusive as the experiment may be considered unphysiologic.

INTESTINAL ABSORPTION

More doubt has been cast on the role of the leukocytes in absorption of fat from the intestine. Leach claims that neither the lymphocytes or eosinophils of the intestinal mucosa contain fat particles. Too, while the phagocytic cells do contain fat granules, available evidence indicates that they are migrating towards and not from the intestinal lumen.

Various workers have tried to disprove the theory of Verzar that bile salts aid in fat absorption by rendering the fatty acids more diffusible. These investigators claim that the higher unsaturated fatty acids, when combined with bile salts, do not diffuse through artificial membranes. Utilization of such membranes makes this very unsubstantial evidence, however.

Sinclair and others have demonstrated a rapid turnover of phospholipids in the intestinal mucosa of fed animals. Radioactive "labeled" phosphorus was used in these experiments. It is not settled whether this rapid turnover indicates that the phospholipids are intermediary products in the resynthesis of absorbed fatty acids and glycerol, or whether phospholipids are synthesized in the intestinal mucosa and distributed to the rest of the body through the blood stream. In favor of the former contention is the fact that a phospholipidemia follows the feeding of fats. However, Hevesy and Lundsgaard, by feeding olive oil and "labeled" phosphorus to animals were unable to show that the increased phospholipids of the blood contained the "labeled" phosphorus.

Frazer has presented evidence to show that that portion of the dietary fat which is hydrolyzed forms soluble complexes with bile salts and is absorbed into the portal circulation to be stored in the liver, while the remainder of the dietary fat is absorbed without hydrolysis into the lymphatic system and is discharged into the general circulation to be stored in the fat depots.

It is a well established fact that patients with sprue, non-tropical sprue, and celiac disease do not show a normal rise in blood sugar or blood fats following the ingestion of glucose or a fatty meal respectively. The administration of liver extract improves both the diseases and the impaired absorption. Castle et al. believe the active principle to be the antipernicious anemia principle. Verzar believes that sprue is an adrenocortical insufficiency rather than a dietary deficiency; thus the liver extract is potent because it provides flavin phosphoric acid, which supposedly the body is unable to synthesize in the absence of the adrenocortical hormone.

There is a selective absorption of sugars in rats; glucose is absorbed 30 per cent

faster in the upper part of the intestine than the lower, while xylose, which is not selectively absorbed, is absorbed at the same rate along the entire small intestine. This selective absorption of glucose is specifically inhibited at lower temperatures. Verzar believes that this selective absorption is dependent on the presence of the adrenals, as their removal destroys the differential absorption rates for upper and lower segments of the small intestine. Conversely, Deuel et al., using adrenalectomized rats maintained in good health by the administration of salt solution, were unable to detect any disturbance of glucose absorption.

Further study on the humoral control of the movements of the intestinal villi and their role in absorption has been done. It was found that the introduction of hydrochloric acid into the duodenum of a dog stimulates the movements of the jejunal villi, thus augmenting the absorption of glucose by 20 per cent. Glucose under the same conditions was without effect. The humoral nature of the mechanism was demonstrated by cross-circulation experiments with carotid-to-carotid anastomoses. Thus the presence of acid in the donor dog increases the movements of the villi and absorption of glucose in the recipient dog. This active principle is called villikinin.

Evidence has been obtained that indicates the presence of active osmotic work by an unknown mechanism in the process of intestinal absorption. Visscher, Ingraham, et al. report that lower ileum of both anesthetized and unanesthetized dogs does osmotic work against a concentration gradient in absorbing sodium chloride from solutions containing mixtures of sodium chloride and disodic sulfate. Using dogs with Thiry-Vella loops, it was found that there was no chloride impoverishment in a high jejunal loop, but the chloride concentration falls rapidly while the sulfate increases in the ileal segments. The concentration of chlorine in the bowel may fall to as low as 0.5 per cent of the blood level. Thus the total osmotic pressure of intestinal contents reached values a half atmosphere below that of blood plasma although the major solute constituents often showed concentration changes opposite to those expected on the basis of simple diffusion.

Upon studying a man with three feet of small intestine, it was found that absorption of carbohydrate was normal, while 25 per cent of the protein was lost; most of the fat was digested but 45 per cent appeared in the feces as fatty acids which interfered with calcium absorption. More evidence has been assembled supporting the classical concept that protein digestion occurs largely in the gut and very slightly in the blood; absorption of undigested food molecules is decidedly minute.

INTESTINAL SECRETION

Stimulation of the vagi produces secretion from Brunner's glands but not from other portions of the intestine. Ivy et al. were unable to collaborate the experimental evidence that acid in the duodenum leads to the production of a hormone, incretin, which supposedly decreases the fasting blood sugar level. (This work is cited above.)

A hormone, distinct from secretin, which excites the glands of the small intestine, has been described; it is called enterocrinin. Its mode of action is as yet undetermined, but it seems likely that enterocrinin acts directly on the glands of the intestine.

Schiffin and Nasset report that feeding causes a diminution of both enzyme concentration and total enzyme secretion of jejunal and ileal segments. This inhibition is overcome in the jejunum six to seven hours postcibum, but persists in the ileum for at least eight hours. As denervation results in an increase in enzyme concentration as well as total secretion after food, it is likely that the first stage inhibition was nervous in nature. Enterocrinin increases the concentration and quantity of enzyme secretion, but does not affect the pH, chloride, or total CO_2. It has been shown that feeding or denervation augments the response to enterocrinin. Secretin is not effective when administered subcutaneously, but enterocrinin is when given in large doses.

GALLBLADDER

More evidence has been accumulated to show that the main function of the gallbladder is to store bile. Morphine delays gallbladder evacuation, presumably by its property of producing spasm of the sphincter of Oddi.

Evidence has been advanced indicating that there is biliary stasis during the last two trimesters of pregnancy. This is probably due to hypertonicity of the sphincter of Oddi, and may account for the high incidence of gall stones in women who have borne children.

BILE

It is claimed that bile administration increases the output of all the bile fractions. In the human, the increase of the "desoxycholic acid" group was greater than that of the cholic acid group; too, the bile acids conjugated to glycine were greatly increased.

Utilizing a human with a bile fistula, Jacobi et al. observed that a pure carbohydrate or pure fat diet does not augment the bile flow above the fasting level. Bile alone or a mixed diet or a high fat diet were equally effective in increasing bile output; however, a protein diet was twice as effective. Addition of bile to the high fat, pure carbohydrate, or mixed diet augmented the output but did not influence the flow from a high protein diet.

Haney et al., by employing the "bolus" method, found that gallbladder bile or a solution of bile salts markedly increased intestinal propulsion. Too, they found that other constituents of bile (bile pigments, calcium, mucin, or lipoids) had no effect. Thus bile salts may play an important role in the normal regulation of intestinal propulsion.

Further evidence has been presented showing that fatty acids play a more important role in maintaining cholesterol in solution and thereby preventing formation of gall stones than do bile salts or bile acids. Lauric and myristic acids are two to three times better solvents than other fatty acids and were forty to fifty times more effective than bile salts.

Ivy has shown that maximum gallbladder response to cholecystokinin takes place when the intravesical pressure is between five to twelve mm. of bile. Puestow believes that cholecystectomy leads to permanent loss of function of the sphincter of Oddi; still the ductus choledochus becomes dilated.

PANCREAS

It has been concluded that the splanchnic nerves to the pancreas are mixed nerves, containing both cholinergic and adrenergic fibers; the former are excitatory and the latter inhibitory. Ivy and Greengard have purified secretin to such an extent that 0.014 mg. constituted one Ivy dog unit. These coworkers have shown that secretin is a true chologogue for it stimulates the formation of bile by the liver after complete removal of the gastrointestinal tract. Injection of bile into a major pancreatic duct of dogs produces a prompt rise in serum lipase, associated with pancreatitis.

COLON

Larson has pointed out that the large intestine is distinctly a double organ, being made up of essentially the right and left portions. This is substantiated by its embryologic development, anatomic structure, and physiologic functions. Thus the proximal one-half of the colon is derived from the midgut along with that portion of the small bowel distal to the papilla of Vater; that portion of the colon distal to the splenic flexure takes origin from the hind gut.

It is claimed by some that amino acids and higher split products of protein are absorbed from the colon more rapidly than is usually reported. It is generally recognized that colonic contents assume the pH of the blood; thus if water at pH 7.3 were injected into the colon and if 20 minutes later the pH were determined, uncompensated acidosis or alkalosis could be recognized.

In a carefully controlled study of colonic motility in dogs, enemas of soap solution were usually effective in producing defecation. When, however, conditions were not suitable for defecation, the soap enema inhibited colonic motility. Diets low in calcium produce hypotonicity of the colon; diets low in potassium have a similar, but

less marked, effect. It is generally acknowledged that pitocin, pitressin, pituitrin, ephedrine, amyl nitrite, papaverine, and histamine decrease colonic tone and motility, while calcium salts, physostigmine, acetylcholine, and hypertonic sodium chloride increase colonic tone and motility.

It now appears that the bacterial flora can be controlled by dietary measures without inoculation with the desired microorganism. Thus rats can be placed on a meat diet until *L. acidophilus* has disappeared from the stool. However, the subsequent addition of banana powder, apple powder, or raisins restored *L. acidophilus* to the feces.

It is generally believed that smoking increases the motility of the colon and promotes the urge to defecate, and, as a rule, this is true. Thus prohibition of smoking by patients with any tendency toward irritability of the colon should be seriously considered. Thus the activity of the stomach tends to be depressed and that of the colon to be stimulated.

It has been shown that stimulation of the nervi erigentes increases colonic secretion.

SUMMARY

In evaluating the literature it is obvious that some work is more conclusive and more important than other investigations. This work is cited below.

Peptic ulcer: More evidence that hyperacidity is unimportant in the etiology of peptic ulcer has been advanced recently. Extensive investigation of the claims that lesions in the hypothalamus produce peptic ulcer shows this contention to be unwarranted. Noting that peptic ulcers occur only infrequently in pregnancy, workers studied the effect of sex hormones on ulcers and found that antuitrin-S decreases the incidence of ulcers in Mann-Williamson animals. There is an ascorbic acid deficiency associated with peptic ulcers; the significance of this has not been shown.

Intestinal obstruction: Wangensteen et al. have strikingly shown that the mechanical factor of distention is much more important than the "toxic factor" as a cause of death in ileal obstruction.

Salivary secretion: The saliva produced by sympathetic stimulation differs constantly from that produced by chorda stimulation. There is a direct linear relationship between the weight of food eaten and the volume of the resulting salivary secretion.

Stomach: Ivy et al. have shown that gastrectomies in growing puppies produces a faulty ossification or homogenous osteoporosis of bones, even though the diet was adequate.

Gastric secretion: Recent investigators report that the smoking of an ordinary number of cigarets increases the acid output of the fasting stomach most infrequently; in fact, depression of gastric acidity is much more frequent. Thus it seems likely that smoking is not conducive to peptic ulceration.

The ingestion of fat or sugars causes the inhibition of gastric secretion and motility by means of a humoral mechanism. The active principle is a specific duodenal cholane: enterogastrone. It is likely that enterogastrone is excreted through the urine, as numerous investigators have prepared extracts from urine, the chemical and biologic behavior of which resembles that of the duodenal preparation of enterogastrone. These extracts thus inhibit gastric secretion and motility.

The consensus among reliable investigators is that the hormone, gastrin, is not identical with histamine. It is likely that histamine mediates the secretion of the parietal cells: the acetylcholine liberated by the vagus nerve liberates histamine which stimulates the parietal cell.

It has been shown that the "intrinsic" factor of Castle is not elaborated solely by the gastric mucosa, for a potent antipernicious anemia preparation can be made from desiccated duodenal mucosa of hogs. Some claim that the "intrinsic" factor possesses proteolytic activity.

There is considerable disagreement as to whether colloidal aluminum hydroxide exerts a stimulating effect on gastric secretion or not; some claim that it actually inhibits gastric secretion.

Gastric motility: It has been shown that the pyloric antrum, the pyloric sphincter, and the duodenal bulb operate as one functional unit; the sphincter acts to prevent regurgitation to a much greater extent than to regulate evacuation.

Conclusive evidence that fats, products of protein digestion, and carbohydrates (listed in order of inhibitory potency) produce inhibition of gastric motility with a corresponding delay in gastric evacuation has been advanced.

There seems to be lack of agreement between hunger sensations and hunger contractions. Apparently, no causal relationship between blood sugar values and normal hunger periods exists.

Duodenum: The question of whether or not there is a duodenal hormone involved in carbohydrate metabolism is a much debated one. Ivy, Loew, and Gray present evidence which makes the existence of such a hormone extremely doubtful, however.

Intestinal motility: The introduction of dilute hydrochloric acid into the intestine increases the motility of the villi through a hormonal mechanism. The active principle is called "villikinin."

Despite the large number of inhibitory reflexes described, as yet only two motor reflexes are known: the gastro-ileac and the gastro-colic reflexes. Strikingly enough, there is no convincing evidence that the vagi play any part in mediating motor reflexes. The gastro-ileal response seems to be mediated by the enteric intrinsic nerve mechanism.

Intestinal absorption: More doubt has been cast on the role of the leukocytes in the absorption of fat from the intestine. There is a rapid turnover of phospholipids in the intestinal mucosa of fed animals; possibly, these are intermediary products in the resynthesis of absorbed fatty acids and glycerol. Selective absorption of sugars has been demonstrated in rats: glucose is absorbed 30 per cent faster in the upper part of the intestine than the lower, while xylose exhibits no such selective absorption.

Visscher et al. have obtained evidence that indicates the presence of active osmotic work by an unknown mechanism in the process of intestinal absorption.

Intestinal secretion: A hormone, distinct from secretin, which excites the glands of the small intestine, has been described; it is called enterocrinin.

Gallbladder: Evidence has been advanced indicating that there is biliary stasis during the last two trimesters of pregnancy; this may account for the high incidence of gall stones in women who have borne children.

Bile: Further proof has been presented showing that fatty acids play a more important role in maintaining cholesterol in solution and thereby preventing the formation of gall stones than do bile salts or bile acids. Bile salts seem to play a part in the normal regulation of intestinal propulsion.

Colon: It appears that bacterial flora can be controlled by dietary measures without inoculation with the desired microorganism. As a general rule, smoking increases the motility of the colon and promotes the urge to defecate.

It is claimed by some that amino acids and higher split products of protein are absorbed from the colon more rapidly than is usually reported.

CONCLUSION

Because of the vast amount of conflicting experimental work, it is difficult to draw any definite conclusions concerning many phases of gastrointestinal physiology.

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DOCTORS IN THE SERVICE

As one meets, sees and talks with doctors throughout the state, he is impressed with the burden that is being placed on the shoulders of the doctors who have not gone into the military services. Everywhere it is found that as the younger men have gone into the Army or Navy, the older doctors have been taking on additional responsibilities and additional work of all kinds. The doctor in civilian practice will have responsibilities thrown upon his shoulders, not only for the care of the sick civilian population in greater numbers than ever before but also he will have the care of the indigent, leadership in civilian defense and the maintaining of health standards in his community. A further burden that he will be asked to carry, if it becomes organized and functioning, probably will be the rehabilitation of the men who were turned down by the Selective Service on account of physicial defects which are remedial. He will have to play an important role in rehabilitating these individuals. Of course there are certain compensations in this extra work, notably the satisfaction of doing a job well but also because additional income will accrue to the man whose practice is materially increased, which income in goodly part will go to the Government for income tax. However, the added revenues will most certainly not repay a man for the energy which he will have to give, the strain he will be under and the uneven prolonged day's work he will have to carry on for twelve to twenty hours.

It is very satisfactory indeed to observe how willing and anxious the men are in the State of Louisiana under the age of 45, to volunteer and to get into active service and to do something for their country. Almost to a man they seem to be desirous of going and when their applications for active duty are not acted upon sufficiently quickly they seem to resent their being held back. All credit should go to these younger doctors for their desire to serve their country. It will be that their income, in most instances, will be materially reduced and many of them are fearful that on their return to civil life their practices will be gone. This, however, was not the experience in the last war. Also these men are going with a good and brave heart. Service in the Army does not imply freedom from danger and even death. The Medical Corps in the last war suffered a very large number of casualties, larger than any other special service, so that the younger doctor with combat troops going forth to war, risks injuries which may be even fatal. However, as with all young men who are going into

the Army, they are accepting this contingency with the thought that if anything does happen to them they are doing it to preserve a way of living which entitles them, their families and all Americans to freedom of thought and of action.

A SCARCITY OF DRUGS?

The receipt of a notice from the chairman of the Committee of Revision of the U. S. Pharmacopeia relative to a change in the mercury content of blue ointment, ammoniated mercury, white precipitate ointment and mild mercurial ointment, is indicative of what possibly may be a serious shortage in some of the essential therapeutic agents employed by the physician. As a matter of fact already conferences have been held by the National Research Council and others pertinently concerned with the problem as to conserving our supply of essential drugs and medicaments brought from overseas. The mercurial content has been materially reduced in these several ointments in order to preserve the present stock of mercury. The price of bichloride of mercury tablets has jumped by leaps and bounds. It is impossible to purchase these important antiseptic agents at many drugstores.

It is to be wondered just how severely felt will be the shortage in many of our drugs, the supply of which comes from China, India, the East Indies, Italy and other countries from which these can no longer be purchased. Many of the galenicals can be grown in this country. The foxglove grows luxuriantly in all parts of the United States and actually is an extremely pretty garden plant. The deadly nightshade likewise is a most attractive garden flower not widely grown because of its poisonous qualities. The fungus *Claviceps purpurea* is found in the grain fields of North America. Nux vomica comes from India, the seeds of a tree indigenous to that country. Quinine of course comes from the bark of cinchona tree of Java. Peruvian bark represents 90 per cent of the quinine used in this country. Ephedrine occurs in various plants of the genus Ephedra and grows in temperate subtropical areas of Europe, Asia and America but most of the supply of the United States comes from northern China. It would take some years, of course, to obtain a supply of these drugs that come from trees; probably the cinchona tree would grow only in Central America although the other trees can grow in colder latitudes. Gum camphor comes also from camphor trees in the East Indies and could possibly be cultivated in this country.

In so far as the mineral and chemical remedies are concerned, the greater part of these can be obtained from sources which are still open to the United States. Mercury is probably the only element, the supply of which is likely to fail. The supply of arsenic, bismuth, antimony, iodine and other minerals is apparently entirely adequate and there is no visible or likely need to restrict the use of these chemicals. Most of the chemical preparations can and are manufactured in this country. Synthetic preparations of all kinds are being substituted to a great extent for already existing pharmaceutical preparations; they can substitute in most instances effectively for the materials that are scarce or unavailable.

There does exist a very real necessity to conserve alcohol and glycerine. They are used to such an extent in the manufacture of munitions that they are more and more difficult to obtain, and most expensive. There is a real shortage of the volatile oils. However, these substances are relatively unessential to the care of the man truly ill. The fact that lavendar can no longer be obtained to scent soaps and that oil of rose will be gone in a short time, is probably of greater importance and interest to the cosmeticians and the female sex of the population than it is to the medical profession.

BREAST PAIN

Nathanson* and his coworkers present a method of treatment of a syndrome which is quite common and for which adequate therapy is not generally applied. In women at the time of the menopause this syndrome is characterized by swelling of the breasts and pain, sometimes with and sometimes

without secretion from the nipple. The syndrome occurs in a very large number of women but often it is not particularly painful and not especially disturbing. Nearly all women have changes in the breasts which they are not aware of in many cases until discovered on routine examination. Others have a great deal of pain and discomfort and because of their fear of cancer will consult a physician.

The authors point out that usually the condition is spoken of as "chronic cystic mastitis" but that it should be more specifically cataloged. The syndrome is presumed to be a sex-hormone imbalance, the nature of which is not entirely clear. If this assumption is true the solution of the problem of treatment resolves itself around the administration of the sex hormone as suggested first by Cutler some eleven years ago. The sex hormone testosterone is given as result of experimental work in animals which shows that this particular hormone not only reduces the secretory activity of the gland but may actually reduce the number of hyperplastic epithelial cells. Presumably also it minimizes the venous congestion and edema observed in the human.

For the last six years these authors have been giving to selected patients testosterone injected intramuscularly, 10 mg. of testosterone propionate in 1 c. c. of sesame oil beginning about two weeks before the anticipated period. In the more severe cases the hormone was given daily throughout the menstrual cycle and in larger doses. They have also administered it in the form of an ointment rubbed into the skin of each breast before retiring.

The patients were carefully studied and those in whom the psychogenic element predominated were excluded, likewise those who had spontaneous remissions after being observed for a period of time. The patients selected for treatment had to have severe discomfort, the presence of marked nodularity of the breast and secretion, failure to control the symptoms with breast support or a pracebo, failure of estrogenic therapy and failure of demonstrable pelvic disease, plus intelligence on her part in order to obtain reliable information. The tables the authors present definitely show the excellent results from this method of treatment. Complete relief or improvement was noted in nine patients who had adenofibrosis. In 21 individuals with nonpuerperal mammary secretion no change was noted in three, complete relief was obtained in 12 instances and there was definite improvement or relief of physical signs in 15. Eighteen of the patients showed relief or improvement in regard to the secretion, although complete relief of the physical signs was obtained in only three.

Nathanson et al. warn against prolonged and continuous treatment, especially with large doses of this substance. They caution concerning the selection of patients, pointing out those with a predominant psychogenic element probably will not have much relief. In those in whom testosterone is given there should be planned periods of treatment succeeded by appropriate rest intervals.

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*Nathanson, I. T., Meigs, J. V., and Parsons, L.: The treatment of mammary pain and secretion with testosterone propionate, New England, J. M., 226:323, 1942.

HOSPITAL STAFF TRANSACTIONS AND CLINICAL MEETINGS

TOURO INFIRMARY

New Orleans

The regular meeting of the Medical Staff of the hospital was held on Wednesday, April 8. The scientific program consisted of the following: Clinico-pathologic Conference by Dr. S. Harvey Colvin; "Gouty Arthritis: Factors in its Diagnosis" by Dr. Daniel M. Kingsley; "Stokes-Adams Syndrome" by Dr. Oscar Blitz.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

TRANSACTIONS OF THE ORLEANS PARISH MEDICAL SOCIETY

During the month of April the Society held one regular scientific meeting. The program was as follows:

a. Case Report—Postpartal Hematoma of the Vulva
   By Drs. John S. Herring and J. P. Michaels.
b. Clinical Status of Typhus Fever at Charity Hospital
   By Dr. Cheney C. Joseph.
c. Blood Volume in Common Clinical Conditions
   By Dr. George R. Meneely.

Reports of Officers and Committees for the first quarter of 1942 were also read at this meeting.

NEWS ITEMS

Drs. H. T. Beacham, W. E. Kittredge, W. A. Reed, R. F. Sharp, E. G. Vickery and H. W. E. Walther attended the eighth annual meeting of the Southeastern Section of the American Urological Association held in Chattanooga, March 19-21. This Association has selected New Orleans as their meeting place in 1943.

Dr. Waldemar R. Metz presented a paper on Some Principles of Plastic Repair at the annual meeting of the Southeastern Surgical Congress held in Atlanta, March 9-11.

At a recent staff meeting of the Mercy Hospital Dr. Lucien Fortier presented a paper on Interesting X-ray Cases. Drs. Edgar Hull and Louis A. Monte presented a paper on Interesting and Unusual Heart Cases at this meeting.

Edwin L. Zander, M. D.,
Secretary.

CALENDAR OF MEETINGS

May 4. Orleans Parish Medical Society Board of Directors, 8 p. m.
May 5. Eye, Ear, Nose and Throat Staff, 8 p. m.
May 6. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Mercy Hospital Staff, 8 p. m.
May 7. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.
Baptist Hospital, Executive Meeting, 8 p. m.
May 11. Orleans Parish Medical Society, Scientific Meeting, 8 p. m.
May 13. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Touro Infirmary Staff, 8 p. m.
Women's Auxiliary, Orleans Parish Medical Society, Orleans Club, 3 p. m.
May 15. I. C. R. R. Hospital Staff, 12:30 p. m.
May 18. Hotel Dieu Staff, 8 p. m.
Clinico-pathologic Conference, Baptist Hospital, 8 p. m.
May 19. Charity Hospital Medical Staff, 8 p. m.
May 20. Clinico-pathologic Conference, Charity Hospital Morgue Amphitheater, 1:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.
Clinico-pathologic Conference, Hotel Dieu, 8:15 p. m.
May 21. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.
May 26. Baptist Hospital Staff, 8 p. m.
May 27. French Hospital Staff, 8 p: m:
Hutchinson Memorial Clinic Staff, 8 p. m.
May 28. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.
May 29. L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital and Dispensary for Women and Children Staff, 8 p. m.

NUTRITION IN CIVILIAN DEFENSE*

Oscar W. Bethea, M. D.
New Orleans

The health of the people is a major problem at all times. Now, it is also a matter of military necessity. Not only must the men in the service and the workmen in the fields and factories be at their best, but the physical fitness, and coincidently the morale of all the others, assume an importance that cannot be over-estimated.

Even before present restrictions in production, transportation and distribution, our diets were unsatisfactory for producing and maintaining the best possible state of well-being. Few indeed suffered from inadequate amounts of carbohydrates, proteins and fats, but the food-intake of the majority was deficient in one or more of such essentials as iron, calcium, phosphorus, and the various vitamins.

McLester once remarked, "It is not merely safety in nutrition that is sought. It is the optimum, and that requires thought." This applies with added force to the present stressful times, as we realize the necessity of rising above our rather mediocre peace-time level. Science has fairly well demonstrated the possibilities that are within our reach.

Sherman and Campbell have stated, "Enrichment of an already adequate diet in respect to its

*Read before general meeting of the Orleans Parish Medical Society on Civilian Defense, March 9, 1942.

calcium, vitamin A or riboflavin content resulted in an improvement in the animal's nutritional well-being, health, and longevity." McCormick has submitted rather convincing evidence of the improvement in sustained physical performance resulting from an increased vitamin intake.

In the study of the food-intake of a large group of Americans, Sherman found 16 per cent definitely deficient in calcium. From an analysis of the data obtained in his study, he also stated that the average diet does not carry an entirely trustworthy surplus of iron.

Some time ago, in collaboration with Bristow, the records of a large group of young women were studied from the standpoint of the hemoglobin content. These individuals averaged from 18 to 25 years of age, and came from several states. Twenty-six per cent were found to have a hemoglobin of 65 per cent or less.

Throughout the centuries of world history, the people have depended largely upon grain for their vitamin B-complex and every country has been well supplied with this valuable material. The Egyptian had his wheat, the American Indian his corn, the Oriental his rice. Many countries had several kinds of grain available. In the latter part of the last century, new processes were developed for improving the appearance of grain products and causing them to keep longer in commerce. These processes, however, removed most of the vitamin content. For example, Cowgill found that white flour contained only about 5½ per cent of the vitamin in the original wheat. He remarks further that American dietaries as a whole are unsatisfactory with respect to their content of vitamin B-1.

Many of us have been inclined to think of vitamin deficiencies in terms of the very poor. Sinclair sums up present medical opinion when he says, "Rich children fed on highly refined foods and stuffed sweets may fare worse than poor children eating scraps."

We must not think of vitamin deficiencies only in terms of definite diseases such as scurvy, rickets, pellagra. These represent but the smallest fraction of the sum total of those who are more or less handicapped. Brown has stated, "Many people are in a condition of marginal or actual vitamin deficiency, and many show no deficiency signs. When the deficiency exists, it is usually a multiple vitamin deficiency." Sinclair also says, "Vitamin deficiencies are rarely single."

By way of suggesting a way out, Youmans recommends "Preference should be given to adequate diet, concentrates or similar preparations of natural foods, and pure vitamins in the order named."

Pure vitamins and the various polyvitamin preparations have served a most useful purpose but their value certainly is being exploited by commercial houses. In 1938 the people of the United States spent over one hundred million dollars for these preparations. Last year it was probably two or three times that amount.

It has been aptly said that the best way to take vitamins is with a knife and fork, and it is this phase of the vitamin problem with which we are concerned in our present program.

Many years ago I realized my inability and my lack of time to tell each patient off-hand which foods to take to secure the best results from the standpoint of general health. I realized even more the inability of the patient to remember such suggestions. To meet my requirements, I arranged a list of about forty of the more desirable foods, had it multigraphed, and have tried to place one of these sheets in the hands of each individual looking to me for health; when desirable, adding verbal instructions and explanations and often making corrections or additions on the sheet given. While I have called it, "Some Vitamin Containing Foods," I have tried to arrange it from the standpoint of a balanced food-intake covering the various essentials. It is not intended as a diet list, but merely as a group of items to be emphasized in the diet. I acknowledge its many shortcomings. Many items rich in various vitamins, calcium, iron, have been omitted. I have been guided largely by palatability, cost, and availability in this market. In some instances, items are included that carry only a small percentage of a desired food-essential but they are of such a type that they are taken in relatively large amounts.

In the present emergency, the Government has undertaken to improve the well being of the population and a major consideration naturally is attention to diet. A National Committee was formed and various local Committees are being organized. We have one in New Orleans. I was appointed by this Society to serve on this Committee as their representative.

In the discussions that developed at our meetings, I mentioned my plan for securing dietary efficiency for those under my care and suggested the program that I am presenting to you tonight. It met with the unanimous approval of the Committee, and I am presenting it as coming from that body.

First: That this Society appoint a Committee to arrange a list of desirable foods that we would recommend as needing emphasis in the food-intake of the people in this community.

Second: That this Committee cooperate with those other agencies concerned with better diet programs in the schools and for welfare work, so that the recommendations that are presented to the public by teachers and welfare workers may come with the added force that it was recommended by their physicians.

Third: That these lists be made available to all physicians at a moderate price or that physicians be permitted to have them printed bearing their own names, and that every physician of this city be urged to get one of these lists into the hands of

everyone looking to him for health, and that the importance of verbal explanations and instructions be emphasized.

Fourth: That effort be made to extend this movement to the State Medical Association.

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LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	

FIFTH DISTRICT MEDICAL SOCIETY

The first meeting of the year was held at the Virginia Hotel in Monroe on Tuesday, April 7. The scientific program was preceded by a dinner at which nearly 150 were in attendance. During the dinner there were half a dozen informal talks, including a very excellent one by Dr. Paul T. Talbot, Secretary of the State Medical Society. Following this the regular formal program was presented, which started with a most heartening and stimulating talk on the affairs of the State Society by Dr. King Rand, President of the organization. Dr. James S. McLester, of Birmingham, then discussed "The Nutrition of the American People in Time of War." Major Frank P. Rizzo of the Selective Service had as a title for his remarks "The Procurement and Assignment of Physicians." He pointed out how doctors were to be procured, how they are to be assigned and then he dwelt on the tentative plans for rehabilitation of the draftees rejected for remedial defects. Dr. John H. Musser completed the program with a discussion of "The Heart in Mid and Late Life."

NEWS ITEMS

The American Public Health Association has prepared and issued two reports which may be of general interest, certainly of special interest to those concerned. The first has to do with the educational qualifications of industrial hygienists, and the second has to do with the educational qualifications of nutritionists in health agencies.

The American Bureau for Medical Aid to China is preparing to spend over a $1,000,000 next year to aid this ally of the United States. Those interested in the work of this organization can send contributions to the National Headquarters, 1790 Broadway, New York, New York.

The Annals of Surgery, the oldest surgical journal in the English language, is now preparing as a gesture of friendship to our Central and South American colleagues an edition published in the Spanish language.

The American Medical Golfing Association will hold its twenty-eighth annual tournament at Seaview Country Club, Atlantic City, on Monday, June 8. Forty prizes will be awarded. All male fellows of the American Medical Association can

belong to this active physio-social organization. Communicate with Dr. Bill Burns, 2020 Olds Tower, Lansing, Michigan, for application blank.

The eighteenth annual meeting of the American Heart Association will be held at Chalfonte-Haddon Hall, Atlantic City, on Friday, June 5. Dr. Paul D. White is president of the organization. The meeting, as has been customary in the last few years, will be divided into two sections, one devoted to cardiac disease, the other to the study of the peripheral circulation.

The Medical Library Association will hold its 44th annual meeting in New Orleans, May 7-9, 1942. The hosts are the Rudolph Matas Medical Library of Tulane University, the Orleans Parish Medical Society Library and the Agramonte Memorial Library of Louisiana State University Medical Center. Hotel headquarters will be at the Jung Hotel. The program will feature tropical medicine and southern medical history. The President of the Association, Miss Mary Louise Marshall will preside.

An intensive course in electrocardiography will be given at the Michael Reese Hospital, Chicago, Illinois, by the Director of Cardiovascular Research, Dr. Louis N. Katz. The course will last for two weeks from August 17 to August 29. This is a course offered principally for the general practitioner.

There is an opening for an eye, ear, nose and throat specialist in Shreveport, La. Further information may be obtained from the office of the Journal.

A physician is wanted for general practice in Bonita, La. For further information communicate with Mr. Arthur Baggett, Baggett Pharmacy, Bonita, La.

United States Public Health Service

Assistant Surgeon James A. Finger has been ordered to the United States Marine Hospital, New Orleans, for duty. Assistant Surgeon Edward P. Cutter has been relieved from duty in New Orleans and ordered to Jacksonville, Florida. Passed Assistant Surgeon Frederick G. Gillick has been relieved from duty in New Orleans and ordered to Jackson, Miss., as State Health Officer. Surgeon David B. Witt has been ordered to the Central Regional Office, Courthouse, Alexandria, La., for duty.

The general oral and pathological examinations (Part II) for all candidates (Groups A and B) will be conducted at Atlantic City, N. J., by the entire Board, from Thursday, June 4, through Tuesday, June 9, 1942, prior to the opening of the annual meeting of the American Medical Association.

Group A, Part II, candidates will be scheduled for examination the first part of the examination period, and Group B, Part II, the latter half. Formal notice of the time and place of these examinations will be sent each candidate several weeks in advance of the examination dates.

DECONTAMINATION OF EYES AFTER EXPOSURE TO LEWISITE AND MUSTARD

Since publication of the Office of Civilian Defense handbooks, "First Aid in the Prevention and Treatment of Chemical Casualties" and "Protection Against Gas," further experience has shown that the 2 per cent solution of hydrogen peroxide recommended for the treatment of eyes following Lewisite burns may be injurious if used undiluted. The Chemical Warfare Service now recommends a single instillation in the eyes of a 0.5 per cent solution of hydrogen peroxide as soon as possible after contamination with Lewisite. This solution may be prepared by diluting one part of a 2 per cent solution with three parts of water, or one part of a 3 per cent solution with five parts of water. The solution usually found in drugstores is the U. S. P. strength of 2.5 to 3.5 per cent hydrogen peroxide. A 0.5 per cent solution of potassium permanganate has also been found effective as an eye instillation following exposure to Lewisite.

In planning decontamination stations, the Medical Division, Office of Civilian Defense, recommends that provision be made near the entrance of the second or shower room for the irrigation of the eyes of contaminated persons. The schematic sketch of a decontamination station in the Office of Civilian Defense publications mentioned above shows the irrigation of eyes in the dressing room, whereas this should be carried out in the second or shower room before the bath is given. Delay until the casualty reaches the dressing room will result in more serious injury to eyes which have been contaminated with mustard or Lewisite.

CONFERENCE OF SOCIAL WORK

The meeting of the National Conference of Social Work and associated groups will be held in New Orleans the week of May 10-16. The organization is made up of the parent association plus innumerable associated groups, ranging from the Salvation Army and the U. S. O., to the National Tuberculosis Association, National Council of Young Men's Christian Association and Birth Control Federation of America. There are five sectional meetings, programs of which will be presented on subjects of interest not only to the social worker but to the physician who comes in contact with the activities of the various groups represented at the meeting.

WAR SESSION MEETING, AMERICAN COLLEGE OF SURGEONS

On March 16, 1942, the American College of Surgeons held a one-day meeting at the Jung Hotel, New Orleans, bringing to the medical professions of both Mississippi and Louisiana the latest methods in treatment and care of present war casualties.

In the beginning of the year the Regents of the College held a meeting to discuss plans for sectional meetings to be held throughout America. A plan was presented to the regents by the associate director, which differed entirely from the previous type of meetings. It was suggested that one-day meetings be held in each state. In a few instances, due to geographical conditions, two states would participate in the program.

The meetings are called the War Sessions of the American College of Surgeons for the medical profession of America. The programs consisted of treatment of all types of war injuries, treatment of war injuries of skull and face, war injuries of chest, the organization and functions of the medical department of the U. S. Army, the organization and function of the medical department of the U. S. Navy, the doctor and the hospital in civilian defense, the procurement and assignment service, treatment of wounds of soft parts, treatment of all types of fractures, treatment of burns, the prevention and treatment of shock, the activities of the American College of Surgeons and their relationship to the defense program, and an excellent resume by Doctor Rudolph Matas of the part Tulane University took in the activities of World War I.

The speakers at this meeting were as follows: from Louisiana—Drs. R. Matas, Dean H. Echols, Alton Ochsner, C. Grenes Cole, Urban Maes, Guy Caldwell, Mims Gage, Neal Owens, Michael DeBakey; from Mississippi—Drs. Augustus Street and Edward C. Parker; from Alabama—Dr. Judson D. Dowling; from the Army and Navy — Colonel Hugh J. Morgan, Major Sam F. Seeley, Army; and Captain Frederick R. Hook, Navy.

By these one-day meetings, the entire medical profession of America has brought to them not only what is expected of them in regard to military associations with the armed forces of the United States, but the most modern and accepted methods of treating war wounds. This enlightenment will give a panoramic view of what is expected of the medical profession in this the greatest endeavor undertaken by the medical profession.

There was a delightful banquet held in the Tulane room, presided over by Dr. Edward Parker, of Gulfport, Miss. At this banquet, the chairman of the Louisiana State Credentials Committee spoke on the activities of the College and its members in the last war, of which membership 99 per cent were on active duty at the time of the armistice. He also outlined the benefits that the public has received from the crusade of the College, especially as regards the standardization of surgical technic, operating room technic, surgical instruments, suture material and gauze dressings, the standardization of hospitals, the creation of surgical residences in a great number of hospitals. In fact, the highest aim and the College's definite policy of constantly elevating the practice of surgery and its specialties has been rewarded by the high type of surgeon and surgical specialists found in almost every hamlet of America. For this the entire medical profession and the laity owe to the College a debt of gratitude that is difficult to repay.

Dr. Matas addressed the entire conclave on the part Tulane took in World War I. A somewhat condensed outline of his speech follows:

At the dinner which closed the one-day session of the American College of Surgeons, held in New Orleans, Monday, March 16, 1942, at the Jung Hotel, Dr. Matas emphasized the value of reviewing the experiences, methods of organization and training which proved of inestimable value during the last war, as a basis for similar organization and centers for training in the present war. Just as it is right to remember the medical men who served their country then, it is hoped that those who serve in the present war will be remembered by future generations.

Dr. Matas outlined the activities of the medical profession in Louisiana during World War I as follows: (1) Medical reserve corps examining boards and medical reservists; (2) Louisiana Council of Medical Defense (a branch of the National Council, with sections devoted to Health, Sanitation and Medical Research); (3) Medical Draft Board of Examiners; (4) committee to standardize medical supplies; (5) Reserve Officers' School of Intensive training in the treatment of fractures, wounds, and other war injuries, New Orleans; (6) Tulane Base Hospital No. 24; (7) Red Cross First Aid Courses; (8) Loyola Base Hospital 102 (under Dr. J. Danna); (9) examining surgeons at Barracks; (10) Tulane University graduates in service (estimated at 578), and undergraduates active soldiers (Camp Martin); (11) hospital staffs; (12) surgeons in the Navy; and in addition, 450-500 nurses served in the Red Cross, Base Hospitals, and Encampments.

Dr. Matas recalled in some detail the Tulane Base Hospital No. 24, which he organized, and the Reserve Officers' School for Intensive Training in War Wounds, Fractures and other war injuries, of which he was the director.

He stressed the importance of the New Orleans Chapter of the American Red Cross under Mrs. George Penrose and Mr. Frank B. Hayne in founding the hospital unit; of Mrs. Tipping as chief organizer of nurses in New Orleans; and Miss Ethel Holmes, acting chief nurse of the unit.

Base Hospital No. 24 cost $100,000, contributed by the citizens of New Orleans, and was stationed at Limoges, France. It was originally planned to provide 500 beds, but when the armistice was

signed, the hospital was caring for as many as 1,741 patients at a time. Its professional personnel included 24 officers, 150 enlisted men and 67 nurses. It left New Orleans under the command of Major Charles McBrayer, U. S. A., with Capt. B. B. Ballanfant, quartermaster. Its medical officers were: Major John B. Elliott, director and chief of the medical section; Major Urban Maes, chief of the surgical section; Capt. John A. Lanford, chief of laboratory; John Smyth, E. D. Fenner, Charles T. Chamberlain, J. T. Halsey, J. D. Weis, I. I. Lemann, Chaille Jamison, Charles Bahn, Harold Kearney, P. King Rand, Paul Lacroix, Muir Bradburn, John Dicks, Warren Scott, Charles K. Wall, E. R. Bowie, Alexander Ficklen; and two dentists, G. E. Sandoz and A. E. Meynier.

The Officers' School for Instruction in War Fractures and other War Wounds, and Collateral Information in Modern War Surgery was established by order of Surgeon General Gorgas, with headquarters at the Charity Hospital, New Orleans, under the direction of Dr. R. Matas, Major M. R. C.

Presentation of standardized surgical material and appliances, and the latest improvements and advances in surgical technic as then developed at the front opened up a large program. A syllabus of some 100 mimeographed pages was prepared and presented in a four weeks' session by Dr. Matas and the following faculty: Drs. F. W. Parham, Hermann B. Gessner, Isidore Cohn, Charles W. Duval, Henry Bayon, W. C. Smith, Amedee Granger, Paul McIlhenny, J. P. Lobenhoffer, Lucian H. Landry, Russell E. Stone, Simon Geismar, I. M. Gage and J. M. Singleton.

Laboratory courses, clinics, and lectures were held at the Charity Hospital, Medical School of Tulane University, and the Touro Infirmary. At the close of each session the attendance and grading of the officers were recorded and reported to the Surgeon General's office. The school continued for eight sessions, from November 5, 1917, to June 30, 1918; and during these eight sessions a total of 138 student officers were in attendance, of whom 11 were majors, 34 captains, 93 lieutenants, coming from 23 military posts in ten states.

The school in New Orleans was one of four similar organizations throughout the country, the others being located in Boston, New York and Chicago. New Orleans was selected as the southern center due to the advantages offered by the large mass of material at the Charity Hospital.

Many expressions of appreciation were received after the war from physicians who had attended the school, and Surgeon General Gorgas, through Colonel Moncrief, expressed his thanks and appreciation.

An interesting feature of the A. C. S. meeting was an exhibit on the activities of the medical profession of Louisiana as described above (1916-1918). It was planned by Dr. Matas, and was held under the auspices of the Medical Library of Tulane, with Miss Marshall, librarian in charge, assisted by Miss Lillian Collens.

The exhibit included framed photographs of the following groups: (1) Executive committee of the General Medical Board of the Council of National Defense, F. F. Simpson, Victor C. Vaughn, W. H. Welch, W. J. Mayo, Gary Grayson, Surgeon General Braisted, Surgeon General Gorgas, Surgeon General Blue, and Franklin Martin; (2) faculty of the School for Intensive Training in Wounds, Fractures and other War Injuries at Charity Hospital, 1917-1918; (3) officer members of each of the school's eight classes; (4) personnel of Tulane Base Hospital No. 24; (5) original letters addressed to Dr. Matas, from King George V, Sir William Osler, Sir Watson Cheyne, Sir Alfred Pierce Gould, Sir D'Arcy Power, in appreciation of the Tulane Base Hospital which he had organized, and from Colonel M. H. Moncrief (representing the Surgeon General) in appreciation of the War School; all from Dr. Matas' collection, and in an excellent state of preservation.

The exhibit also included a large collection of official manuals, textbooks and other literature on the surgical and medical aspects of war, especially prepared for military surgeons of the American, British, French and German armies engaged in World War I, now a part of the College Library; newspaper clippings of 1917-1918, saved by Dr. Matas; and the syllabus used for instruction in fractures, war injuries and war surgery in the classes for intensive war training at Charity Hospital, 1917-1918, prepared by Dr. Matas, by order of the Surgeon General.

MIMS GAGE, M. D.

MERIT SYSTEM EXAMINATIONS

A new series of open competitive examinations for positions in the State Department of Public Welfare, State Board of Health and Department of Labor was announced today by Jack H. Foster, Merit System Supervisor.

A number of varied technical and specialist positions, offering opportunity for rapid advancement are to be filled by examinations which will be given by the Louisiana Merit System Council in the near future, according to Mr. Foster.

The announcement included the following positions and monthly salary ranges: Key punch operator, $90-115; senior key punch operator, $125-150; tabulating equipment operator, $110-150; senior tabulating equipment operator, $150-190; tabulating equipment supervisor, $175-215; statistical clerk, $130-175; statistician, $160-200; senior statistician, $200-250; research analyst, $160-200; senior research analyst, $200-250; chief of research and statistics, $250-300; chief public health statistician, $350-450; senior public health statistician, $250-300; health records advisor, $175-225; health records registrar, $250-300.

It was pointed out by Mr. Foster that in some instances the entrance salaries will be higher than the minimum indicated above.

All qualified residents were urged by Mr. Foster to apply for entrance to the examination and gain a place on the job register.

Applications will be accepted from high school and college seniors, if otherwise qualified, subject to the successful completion of the required high school education prior to July 1, 1942, he said. Examinations will be given some time in May at various points in the state, to be determined later on the basis of the number of applications received. Application forms may be secured by writing the Merit System Supervisor, 631 Main street, Baton Rouge, or from any local United States Employment office, or from the office of any parish department of Public Welfare. Application for entrance to the examination must be made to the Merit System not later than May 5, 1942.

HEALTH OF NEW ORLEANS

The Bureau of the Census, U. S. Department of Commerce, has reported for the week closing March 14 out of the 173 deaths reported in the city, 112 were in the white race, 61 in the negro, and the 11 in infants. This was a very marked increase from the previous week when only 107 of the citizens of the city expired. The following week ending March 21, the number of deaths was still above the three-year average with 164 deaths recorded, divided 97 white, 67 negro, with 13 infants, 10 of whom were negro babies. The figures took a sharp decline the week ending March 28, when only 131 deaths took place in New Orleans. Of those dying 85 were white, 46 negro, and of the 12 infant deaths, contrary to the usual finding 10 were in white babies. For the first week in April, which ended on the fourth, 140 deaths were proportionately 90 white and 50 negro. Thirteen in infants under one year of age were divided about as equally between the two races as it is possible to divide an uneven number.

INFECTIOUS DISEASES IN LOUISIANA

The morbidity report of the Section of Epidemiology of the Louisiana State Board of Health shows that for the week ending March 14 syphilis customarily led all reportable diseases with 296 cases listed this particular week, 28 of which, however, arose in army camps. The very large number of gonorrhea cases that were reported were also due to the fact that 146 of these cases were reported from the army. There was an epidemic of measles throughout the state with 136 cases recorded, followed in proportion of frequency and numbers greater than 10 by the following diseases, pneumonia, 51 cases; mumps, 40; influenza, 27; chickenpox, 25; tuberculosis, 19; and whooping cough, 10. This latter does not represent the number of cases by any means, because it is impossible to conceive of nine of the ten cases coming from one parish, that of Lafourche. Whooping cough was too prevalent and widespread a disease to be recorded solely in one section of the state. Evidently the doctors of Lafourche are more punctilious in reporting whooping cough than doctors elsewhere in Louisiana. For the week ending March 21, 428 cases of syphilis were followed in numerical degression by 188 of measles, 47 of gonorrhea, 36 of chickenpox, 35 of mumps, 30 each of pneumonia and pulmonary tuberculosis, 26 of whooping cough. Two cases of poliomyelitis were reported this week, one from Jefferson and one from Tensas parish. Whooping cough this week was scattered throughout the state, six parishes reporting having this disease among their children. For the week which came to a close March 28, there were listed 232 cases of syphilis, 133 of gonorrhea, 100 of measles, 37 of mumps, 27 of pulmonary tuberculosis, 23 of chickenpox, 22 of chancroid, 16 of pneumonia, and 10 of diphtheria. There were eight cases of typhus fever listed this week, seven of which came from Madison Parish. All the reportable diseases were materially increased in number by the cases coming from army camps.

The epidemic of measles was well on its way to a peak. For the week of April 4 there were more cases of measles reported than any of the other reportable disorders. Of the 292 cases of measles, 61 were in the army camps. Other diseases in figures greater than 10 included 141 cases of syphilis, 48 of mumps, 23 of gonorrhea, 22 of chickenpox, 17 of whooping cough, 15 of pneumonia, 14 of pulmonary tuberculosis. There were two additional cases of typhus fever reported from Madison Parish, and of the 17 cases of whooping cough 12 developed in St. Mary Parish. This latter parish might be compared favorably to Lafourche in attentiveness of physicians in their report of the cases.

DR. S. L. SHAW

1874-1941

At 8 o'clock on the morning of September 30, 1941, Dr. S. L. Shaw suddenly passed away at his home near Clinton. With medicine bag in hand he had started for his car but got only as far as the edge of the front porch when suddenly he dropped into a convenient chair, and was gone even before his nearby wife could reach him. Stricken falling in line of duty.

Dr. Shaw was born August 14, 1874, at Summer Hill, Illinois. He graduated in medicine (1909) from the St. Louis College of Physicians and Surgeons, St. Louis, Missouri. He began the practice of medicine in Clinton, La., in 1925, and was active in his profession up to his last moments. He had been president of the Bi-Parish Medical Society, and was the coroner of East Feliciana Parish at the time of his death.

It was a custom of Dr. Shaw and his lovely wife to entertain the Medical Society every year at the log cabin, near their home. Here they served a barbecue dinner, a real feast, and these occasions will never be forgotten by those persons who were present.

Dr. Shaw was a very unique and interesting personality, broad minded, kindhearted and charitable towards people of all classes, races and creeds. He kept posted in the progress of medicine. He rarely missed being present at the society meetings, and loved to take part in the discussion of the subjects presented. His ideas were good, and always expressed in clear concise language, right to the point. We shall miss him.

Therefore, be it resolved by the Society that we feel keenly the loss of this valuable member, our brother and friend.

Resolved further that we extend our sympathies to his bereaved wife, also to his son and daughter.

Resolved that a copy of this simple tribute be sent to his family; also to the local papers, and to the New Orleans Medical and Surgical Journal for publication.

E. M. ROBARDS, M. D.
E. M. TOLER, M. D.
W. J. ROBERTS, M. D.

BOOK REVIEWS

Diagnosis, Prevention and Treatment of Tropical Diseases: By E. R. Stitt, Rear Admiral, U. S. Navy. Rev., rewritten and ed. by Richard P. Strong. Philadelphia, Blakiston Company, 1942. Pp. 1,747. Price $21.00.

This work might well be called a system of tropical medicine and hygiene. The modest volume hitherto known as Stitt's Tropical Diseases has become a two volume work, full of useful, well-sifted information. It should prove indispensable to physicians in the tropics, a valuable handbook to every practitioner in the medical field, and a useful work of reference for medical students. It appears most opportunely because it is quite obvious that physicians in the future will have to pay more attention to diseases of warm climates than at any time since the expansion of American interest brought about by the Spanish-American War. In the new volumes the public health aspects of diseases are given special emphasis.

It is difficult to restrict a work to diseases found only in warm climates, nor have the authors tried to do this.

Numerous distinguished authorities are mentioned from whom assistance was received in the preparation of the work. There are many new illustrations in the volumes as well as some reproductions. At the end of the various subjects will be found references that should prove most valuable.

Volume I is made up of: section I, dealing with protozoal diseases and section II, dealing with bacterial diseases.

Volume II is made up of six sections as follows: section III, filterable viruses, rickettsiae, and allied organisms; section IV, nutritional disorders; section V, diseases not otherwise classified. (Acute effects of heat, tropical ulcer, granuloma venereum, climatic bubo and a number of rare conditions or those of doubtful origin); section VI, fungi and poisonous plants; section VII, animal parasites, including helmiths, injurious arthropods, poisonous fish, snakes and coelenterates; section VIII, devoted to general and statistical information.

The appendix is divided into three sections: I, index of clinical signs; II, laboratory procedures indexed by disease; III, subdivided into: tropical hygiene, personal hygiene, disinfectants and disinfestants.

A few items of special interest are noted here: It is fitting that malaria, the most important of tropical diseases, occupies more space than any other subject — 135 pages. It is encouraging to learn that *Anopheles gambiae,* which was introduced into Brazil a few years ago is being held in check and there seems to be hope of eradication of this menacing carrier of malaria.

Pinta is looked upon as a treponema infection, in keeping with the recent views of certain Latin American workers, the organism being very similar to *Treponema pallidum.*

Carriers of *Endamoeba histolytica* are divided into: (a) contact (asymptomatic) and (b) convalescent (recovered), and the need of methods of distinguishing pathogenic and non-pathogenic ameba is mentioned.

Sulfanilyl-guanadine is especially recommended for the treatment of bacillary dysentery. Stress is laid on undulant fever as an occupational disease, occurring in shepherds, farmers, farm laborers, men employed in slaughter houses.

It is refreshing to find that the authors recognize that the focus of leprosy in Minnesota has become non-existent.

The chapter on yellow fever, prepared by A. W. Sellards, illustrates very well the stress laid on history, transmission, immunology and prophylaxis. Only eight of thirty-one pages are given over to clinical manifestations, symptoms, diagnosis, pathology and treatment.

Epidemic and endemic typhus are discussed under different headings which simplifies these sub-

jects from the viewpoints of transmission and prevention.

One is pleased to find a textbook which recognizes that the virus of Rocky Mountain spotted fever has thus far not been found in any rodent infected in nature, a contrary statement being the rule.

George C. Shattuck summarizes nutritional diseases. In the discussion of pellagra it is stated that "As late as 1930 some still believed pellagra to be an infectious disease." The prevalence of pellagra on a large scale is recognized as an economic problem as well as one due to the lack of education in the essentials of nutrition.

Tropical ulcer (tropical sloughing phagedena) is regarded as a clinical entity, ulcerations of known etiology having been excluded.

Under the title climatic bubo, the condition usually designated as lymphogranuloma inguinale and more recently often as lymphopathia venereum, is discussed with a full consideration of the Frei test, pointing out especially possible fallacies in the test.

The authors express very conservative views in relation to the use of biological products against certain bacterial diseases. They do not advocate vaccination against bacillary dysentery, and point out possible fallacies in the favorable reports on anti-cholera vaccination and are lukewarm in respect to anti-plague vaccination. Anti-dysenteric serum is regarded as of little value and the evidence with respect to the curative value or lack of value of anti-plague serum is set forth.

One turns with interest to controversial subjects on which the views of the authors are expressed: The evidence makes it clear that the disputed question as to whether malaria gives at times a positive serologic test for syphilis must be answered in the affirmative.

Stress is laid on the importance of quinine prophylaxis of malaria in military preventive medicine and it is emphasized that this treatment does not prevent infection, only the development of symptoms, and indeed that even in this it is not uniformly successful.

The question as to the relation between yaws and syphilis is considered conservatively with the conclusion that "Yaws is a modified virus of syphilis—a less virulent one, producing a disease which has been modified through many years of successive passage of the virus through the epidermis of black skinned races, by the habits of these people, and by the climatic and hygienic conditions under which they live."

Food handlers are regarded as of not much importance in the transmission of amebic dysentery—but it is urged that carriers of the infection should not be employed as food handlers until well treated. The identity of alastrim and smallpox is accepted without qualification.

Having mentioned with approval all of the foregoing, it may be appropriate to call attention to a few features that may be the subjects of differences of opinion. In the discussion of plague Verjbitski's work is not mentioned but there is a synopsis of much of the work of the Advisory Committee appointed by the Secretary of State for India, the Royal Society and the Lister Institute, generally referred to as the Indian Plague Commission.

The authors, together with many others, are more impressed with the similarity between tularemia and plague than is believed warranted. Under the discussion of the diagnosis of undulant fever, *Bacterium tularense* is stated to be much more closely allied to *Bacillus pestis* than to brucella; in fact it is not closely related to either. On page 1673 in the Index of Clinical Diagnosis we find the following: "Tularaemia is a plague like disease occurring in man following contact with rabbits or other rodents." Plague and tularemia in man have but little in common. The statement also omits sources of infection other than rabbits of which ticks and biting flies are important. The symptomatology of the two diseases is very different and the similarity from the gross pathology viewpoint relates only to certain laboratory animals—where of course, the resemblance almost defies differentiation.

The portions of the work dealing with the general aspects of tropical disease and disease prevention are notably good: In the section on Problems of Medical Practice in the Tropics certain general considerations are covered and the authors stress the cosmopolitan nature of many diseases seen in warm climates. This section has a discussion of anemia as well as of several other subjects that would be useful as part of a handbook of general medical practice.

In the appendix, under "Tropical Hygiene" will be found useful information on subjects running from "climate influence" to "control of native tropical races," and including such diverse subjects as laundering and vaccinations.

Viewed as a whole these volumes may be said to contain the most modern and comprehensive discussion of tropical medicine in English, with very little that may justly be criticized and much to be praised. Every special student of tropical diseases and every physician practicing in warm climates will feel indebted to Admiral Stitt and Dr. Strong.

G. W. McCoy, M. D.

Encephalitis, a Clinical Study: By Josephine B. Neal, A. B., M. D., Sc. D., F. A. C. P., and Collaborators. New York, Grune & Stratton, 1942. Pp. 563. Price $6.75.

Epidemic encephalitis is a major neuropsychiatric problem. This disease was probably first recorded in its present epidemic form in Rumania in 1915. However, historical studies indicate that outbreaks of acute encephalitis have occurred throughout the centuries. It is believed that the

last epidemic may have been a product of the impact of the first World War, due perhaps to its depleting and nerve-wracking effects. If so, it is possible that the strain, privations and nutritional deficiencies engendered by the present war may prepare the way for another epidemic.

Under a grant from The William J. Matheson Commission for Encephalitis Research, Neal, with her distinguished collaborators, has made an exhaustive and comprehensive study of encephalitis since 1918. Neal is a brilliant scientist and a gifted writer, and the results of her studies are set forth in this colorful, practical, splendidly balanced and complete work, which abounds in fascinating detail, bears evidence of careful scholarship, and brings the knowledge of encephalitis quite up to date.

It is believed that the weight of opinion is against the possibility of direct relationship between influenza and epidemic encephalitis. Despite twenty-five years of intensive research no final conclusions have been reached in regard to a specific etiologic agent. But it has now been definitely ascertained that there are four types of encephalitis caused by viruses.

There is no unanimity of opinion as to any invariably efficacious plan of treatment. Since no causative factor has been found, the treatment of encephalitis is largely empiric.

It is not often that you see a better book than this, and one cannot review it without being aware of the fact that a new and ever widening era has opened in neuropsychiatry.

C. P. MAY, M. D.

Clinical Roentgenology of Pregnancy: By William Snow, M. D. Springfield, Illinois, Charles C. Thomas, 1942. Pp. 178 with 119 illustrations. Price, $4.50.

This volume is intended as a working manual and a practical reference covering the methods of x-ray pelvimetry and cephalometry employed by the author at the Bronx Hospital and the Harlem Hospital, New York City. The subject matter has been divided into two parts: one, the study of the maternal pelvis and the fetus, and two, the study of the soft structures of pregnancy.

Chapter I consists of a general discussion of the use of the x-rays in pregnancy and includes brief discussions of various methods of pelvimetry and cephalometry employed at the present time. The principles of the author's method are also discussed. The advantages of obtaining the pelvic diameters in terms of linear measurements are considered briefly. The corrected lengths of the diameters are obtained by the use of a slide rule or by a graph devised by the author. The advantages claimed for this method are: (1) accuracy within 5 mm.; (2) rapidity of determining the entire set of measurements in a few minutes; (3) simplicity; and (4) linear measurements.

In Chapter II, the positioning of the patient and the technic of x-ray exposures are considered in detail. Chapter III includes a description of the necessary computations. The technic of measuring the fetal skull consists of a modification of the Ball method of determining the perimeter and of the McNeill method of obtaining the diameter of the fetal skull. By the use of a table or chart, it is then possible to obtain the approximate age and weight of the fetus.

Chapter IV is devoted to a consideration of the pelvic shape in relation to its measurements. The classification of Caldwell, Moloy, Swenson and their coworkers are discussed and an effort is made to classify pelves based on measurements.

Chapter V considers the relationship of the normal and the abnormal fetus to the planes of the pelvis. Chapter VI is concerned with a discussion of the visualization of the soft tissues in pregnancy. Chapter VII consists of a series of twenty-two case reports with reproductions of roentgenograms in each case.

In general, this book is well written and beautifully illustrated. A short bibliography of the most important contributions to the subject and an index are included.

J. N. ANE, M. D.

Synopsis of Allergy: By H. L. Alexander, A. B., M. D. St. Louis, C. V. Mosby Co., 1941. Pp. 246. Price, $3.00.

As indicated in the preface, the purpose of this book is to present the subject of allergy in present day thoughts. Dr. Alexander has succeeded admirably in carrying out his intention. This small volume is a compact but inclusive treatment of the subject. Atopy, hay fever, bronchial asthma, atopic rhinitis, allergic dermatoses, physical allergy, and bacterial allergy are among the subjects succinctly discussed. Much controversial matter has been omitted. There is also an appendix which contains formulae for various extracting fluids currently in vogue, directions for preparation of extracts, the various physical and chemical methods used to standardize extracts, as well as lists of household materials and foods, and the possible allergens which they may contain. The technics of skin testing are also described. An unusual and extremely useful feature of Dr. Alexander's book is the tabulations outlining desensitization schedules. This book is an excellent summary of present day allergic practice.

BERNARD G. EFRON, M. D.

Neuroanatomy: By Fred A. Mettler, A. M., M. D. Ph. D. St. Louis, The C. V. Mosby Company, 1942. Pp. 476, figs. 337. Price, $7.50.

"The present text has been written to meet the needs of the medical student beginning instruction in neuroanatomy and to prepare him for the demands which will later be laid upon him in his clinical training." [From preface.] Opinions as

to how well that aim has been realized naturally will vary among those who teach neuroanatomy, and while it may be unfair to pass judgment at all without having first actually tried out the book on at least one class, its outstanding merits and apparent faults may be enumerated:

(1) Illustrations form an all-important feature of any anatomy text, since on so many points pictorial representation is more effective than words. The figures in Mettler are refreshingly new, beautifully executed and in the main well planned for their intended purpose. (2) Organization of subject matter is an especially critical element in the presentation of neuroanatomy, owing to the intricacy of factual material and the need of proper development of the background for correlation of structure and function. In its general outline this book has a logical organization, with the descriptions of gross anatomy preceding the treatment of minute structures and functional anatomy. Closer reading, however, discloses spots in which the planning may not be such as to lead the student through progressive steps into an appreciation of functional pathways. The lapses may be filled in by the instructor, of course, but it is unfortunate that discussions of functional pathways are so disjointed. (3) Rigorous selection of subject matter is necessary in a text for beginners, who are expected to gain working knowledge of neuroanatomy in a very brief period. The present text devotes much space to some matters of descriptive anatomy which have questionable place in an elementary text, and without indication of their relative unimportance. Except for this, the content of the book is distinctly practical. (4) The legitimate demands imposed on a medical student for the acquirement of an extensive and new vocabulary are already so great that he is in no position to tolerate extravagance in textbook terminology. The author appears to have overemphasized terminology. Even when the English form of a name has gained well-nigh universal use to the exclusion of its technical equivalent, both are listed and often together with some proper name which may have been long obsolete. Then again, in some instances where technical names have been long ingrained in usage, the author gives precedence to the unfamiliar English literal translation. Thus, for example, "slender fasciculus" is first used as the name of the fasciculus gracilis, the pons becomes "bridge," and so on. Why should the medical student be burdened with synonyms except in those instances where two or more names for the same structure are in actual common use?

HAROLD CUMMINS, Ph. D.

A Textbook of Neuro-anatomy: By Albert Kuntz, Ph. D., M. D. Philadelphia, Lea & Febiger, 1942, Pp. 518, fig. 307. Price, $6.00

First published in 1931, this text is already widely known as a sound work covering its field. The content and organization are indicated by the following sequence of chapter topics: Evolution and comparative anatomy; embryology; topography of the central nervous system; meninges and cerebrospinal fluid; interstitial tissue of the central nervous system; myelinization; nervous integration; cerebrospinal nerves; internal structure of spinal cord; spinal conduction pathways; medulla; pons; mesencephalon; long conduction pathways; central connections of nerves III-XII; cerebellum; visual apparatus; autonomic system; cerebral hemispheres, general morphology; olfactory apparatus; cerebral hemispheres, internal structure; cerebral cortex; functions of cerebral cortex; laboratory outline. For this revised edition the text has been partly rewritten and some of the original illustrations replaced by improved figures. The newly added material mainly concerns thalamic connections, the hypothalamic nuclei and details of the cerebral cortex.

HAROLD CUMMINS, Ph. D.

PUBLICATIONS RECEIVED

C. V. Mosby Company, St. Louis: Synopsis of the Preparation and Aftercare of Surgical Patients, by Hugh C. Ilgenfritz, A. B., M. D., and Rawley M. Penick, Jr., Ph. B., M. D., F. A. C. S., with foreword by Urban Maes, M. D., D. Sc., F. A. C. S.

Stanford University Press, Stanford University, California: Lane Medical Lectures: The Lymphatic System, by Cecil K. Drinker, M. D.

Lea & Febiger, Philadelphia: Endocrinology, by August A. Werner, M. D., F. A. C. P.

Grune & Stratton, New York: The Clinical Application of the Rorschach Test, by Ruth Bochner, M. A., and Florence Halpern, M. A. Nephritis, by Leopold Lichwitz, M. D.

The Commonwealth Fund, New York: Neural Mechanisms in Poliomyelitis, by Howard A. Howe, M. D., and David Bodian, Ph. D., M. D. Psychiatry in Medical Education, by Franklin G. Ebaugh, M. D., and Charles A. Rymer, M. D.

The Colt Press, Paterson, New Jersey: Medical State and National Board Summary, by William H. Kupper, M. D.